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THE LEWIS BLOOD ANTIGENS AND ANTIBODIES.

By R. T. SIMMONS AND RACHEL JAKOBOWICZ,

From the Commonwealth Serum Laboratories, the Australian Red Cross Blood Transfusion Service (Victorian Division) and the Queen Victoria Hospital, Melbourne.

THIS communication is presented in the hope that the observations made over a considerable period may throw further light on the Lewis blood group antigens and antibodies.

Mourant (1946) reported the discovery of a "new" blood group which he designated Lewis. The Lewis antigen was found to be present in about 25% of 96 English blood samples tested, and was shown to be independent of the A B O, M-N, Rh, P, Lutheran and Kell systems.

Andresen (1947) described what he thought was a "new" antigen and antibody, but they were subsequently proved to be identical with the Lewis group of Mourant. Andresen fortunately used the letter L to designate his blood group system. He showed that 21% of adult Danish blood samples were L+. It was also found that L+ reactions in young children varied according to age: of children to the age of three months 79% were L+, of those aged four to six months 73% were L+, of those aged seven to nine months 36% were L+, and in a group aged ten to twelve months the L+ percentage fell to 29. He showed that L– parents might have L+ children and suggested that two genes L and l were involved, L representing the character demonstrated, and l its absence. In adults the LL homozygotes gave the L+ reaction, while in young children the heterozygotes Ll as well as LL gave the L+ reaction. In adults Ll and ll persons were non-reactors—that is, L–. This was explained on the grounds that l in adults was a dominant character. It was suggested that l progressively became dominant over L during the first twelve months of life.

Andresen (1948) described a second antibody related to the Lewis system, which he called anti-L_2. Subsequently, Mourant's Lewis antibody and Andresen's anti-L_2 have been designated anti-Le^a and anti-Le^b respectively. Andresen showed that the two antibodies when used to test blood samples of groups O and A_2 behaved in an almost completely antithetical manner. However, when anti-Le^b was used to test blood samples of group A_1, it did not react with 70%, as with samples of O and A_2, but with only 42%. He suggested that this unexpected finding could be explained by an effect known in genetics as epitasy—a term used to describe the "suppression" of one inherited characteristic by another. In this case it would appear that the A_1 factor makes it sometimes impossible to demonstrate the Le^b factor in a proportion of the cells tested. The proportion of non-reacting cells suggests that the suppression occurs only as a "double-dose" effect in homozygous A_1A_1 cells. Of the 238 group O cells tested, all but 6% reacted with either anti-Le^a or anti-Le^b. These non-reactors are now referred to as type Le(a–b–), and the two reacting types as Le(a+b–) and Le(a–b+). The percentages found in the two latter types were 20 and 74 respectively.

Andresen and Jordal (1949) described a further antibody found in the serum of an A_1 Le(a–b–) mother, which they called anti-X. This serum contained an anti-Le^a agglutinin and an "incomplete" antibody, and it reacted with about 90% of all blood samples when the test cells were suspended in their own plasma, or in albumin. They tested this serum with some 300 blood samples. All anti-X negative cells were found to be Le(a–b–), and in the case of groups O and A_2, all Le(a–b–) cells were also anti-X negative.

They found that anti-X serum reacted only at room-temperature, whereas their anti-Le^a serum would react at 37° C. Anti-X serum in addition differed from their anti-Le^b serum in that it gave (i) the same percentage of reactors with groups O and A_1 cells (that is, there was no epitasy), and (ii) approximately the same percentage of reactors in adults and children. Absorption with Le(a+b–) cells removed all the agglutinins from both anti-Le^a and anti-X serum. Andresen and Jordal suggested that X is an inherited factor, that a corresponding allele x must also exist, and further, that the homozygote XX inhibits the development of phenotypes of the Lewis system.

Andresen (1949) in a personal communication informed us that two further examples of anti-X serum had been sent to him, and that these two antisera were of the agglutinating type, in that they gave 90% reactors with cells suspended in saline.

The literature relative to the Lewis blood groups has been recently reviewed by Simmons, Semple and Graydon (1950). These authors tested 1123 blood samples from unrelated white Australians, and found that 26·5% were Le^a positive. In a total of 203 corresponding blood and saliva samples tested, there was correlation in 202 between the Lewis, Le(a+) type and A, B or H secretor status. This correlation is similar to that first shown by Grubb (1948), and by others.

The foregoing brief summary of the activities of the antibodies Le^a, Le^b and X as previously described, has been given as a basis for comparison with three antibodies of several of these types studied by us.

Materials and Methods.

The three antibodies referred to are as follows:

1. Anti-Le^a (Mrs. M.P.). This group O serum, which is a potent example of its type, was found in 1946 and described by Jakobowicz, Simmons and Bryce (1947). After storage for four years at 5° C. in a sterile condition, it has shown no detectable loss of activity. It is active both at room temperature (20° C.) and at 37° C., but its reactivity is far stronger at 20° C. All tests performed with this serum, as with the other two to be described, have been made by a slide technique. To one drop of the undiluted serum on a clear glass slide was added one drop of a 10% cell suspension in glucose-citrate solution (pH 7·4), and, after thorough mixing, the slide was placed in a moist chamber at 20° C. The results were usually read after the slides had stood for fifteen minutes. These slides, like similar slides prepared for testing for A B O groups, M-N, Rh types *et cetera*, should not be rotated, but should be tilted back and forth very gently two or three times while being examined. The Le^a positive reactors gave "++++" or complete agglutination, and these cells were presumably of genotype Le^{a}Le^a. It is thought that persons whose genotype is Le^{a}Le^b give the same phenotype reactions as those whose genotype is Le^{b}Le^b. The anti-A and anti-B activity of the serum could be neutralized by dried A and B group-specific substances of animal origin. Human A and B substances (dried or wet) neutralized the Lewis as well as the anti-A and anti-B activity. This Le^a serum was detected after a blood transfusion reaction.

2. Anti-Le^b (Mrs. Mal.). This serum was from an Le(a–b–) woman of group AB, who at delivery of a healthy full-term baby had an irregular agglutinin which exhibited the characteristics of the anti-Le^b serum of Andresen; that is, with Le (a–) cells of group O and A_2 cells it gave strong agglutination at 20° C. When tested with Le(a–) cells of group A_1 it reacted well with some, but failed to react with others. It failed to react with cells of any A B O group at 37° C. This agglutinin could not be demonstrated in the cord blood, which furthermore gave a negative response to the Coombs test.

Seven days after the patient's confinement, when a large blood donation was obtained, the characteristics of this serum had changed. Owing to either specific or non-specific stimulus, the serum had greatly increased in titre, and it now reacted with all of a small panel of Le(a–) cells irrespective of their A B O blood group. Three strengths of reactions were observed ("++++", "+++" and "++"), and the weakest were those with Le(a–) A_1 cells which had not been agglutinated by the serum sample obtained at confinement. Further, the serum was now found to be capable of reacting at 37° C. with all the previously used Le(a–) cells, again in graded strengths. Thus it appears that the typical anti-Le^b serum of Andresen showing epitasy and not reacting at 37° C., which was present in a patient at confinement, is either a natural agglutinin or is possibly one in the early stages of antibody development, and that the same patient may go on to produce a stronger antibody without the complete epitasy effect, and capable of reacting at 37° C. in the same way as examples of anti-Le^a serum. Because of the graded reactions obtained with cells of different A B O blood groups, it was decided to perform all subsequent anti-Le^b tests at 20° C. as for anti-Le^a, but to read the results after the slides had stood for sixty minutes at 20° C. In practice this procedure gave complete satisfaction. With tests on group O cells, however, it was possible to read both anti-Le^a and anti-Le^b results after fifteen minutes at 20° C., and almost complete antithetical results were obtained.

3. Anti-X (Mrs. Arm.). This serum of group O in an Le(a–) pregnant woman was first detected and investigated in September, 1949, after a reaction to a transfusion with Le(a+) blood. Its specificity was strongly anti-Le^a, but weak reactions were also detected with most Le(a–) cells at 20° C., which suggested that a second but weaker antibody was also present. The weak type of reaction was evident at 20° C., but not at 37° C., at which temperature the anti-Le^a component was active. Of 82 samples of group O cells tested at this time with undiluted serum, 24 known Le(a+) cell samples were found to give strong reactions, 53 Le(a–) to give weak reactions and five Le(a–) to give no reactions; in all, 94% of the test cell samples reacted with serum "Arm". It was thought then that this serum was similar to the anti-X serum of Andresen, but of the agglutinating type. It was decided to follow the antibody activity of Mrs. Arm.'s serum over a period of months. The baby was normal in all respects. Some months after confinement a large blood donation was obtained from the patient. It is this serum specimen which has been used in all subsequent tests. The titres obtained with Le(a+) and Le(b+) group O cells at different temperatures are shown in Table I.

Our observations have shown that only the anti-Le^a portion of this anti-X serum was of sufficient strength to react at 37° C., and to cause a transfusion reaction, and further, that the anti-Le^b component had increased in strength after the patient's confinement, so that while originally it acted only when undiluted, the last sample obtained was active at a dilution of 1:5. The increase in titre was possibly due to the transfusion reaction, or to sensitization by the fœtal cells.

It has been shown that a serum possessing the typical anti-Le^b of Andresen showing epitasy and not reacting at 37° C. has in a patient subjected to further antigenic stimulus progressed to one reacting at 37° C., and without the complete epitasy effect; also, that the individual components of a serum similar to the anti-X serum of Andresen and Jordal have each individually increased in titre in a patient subjected to further stimulus. These observations strongly suggest that the serum regarded by us as anti-X is in actual fact anti-Le^a + Le^b. This suggestion, if correct, therefore throws some doubt on the statement of Andresen and Jordal that anti-X is a separate entity within the Lewis blood group depending on an inherited factor X.

It is considered also that the trace reactions observed by most workers when using anti-Le^a serum with cells other than the strongly reacting Le^{a}Le^a genotype may have been due to slight amounts of anti-Le^b also present in the testing serum.

As our anti-X serum was of group O, neutralization of the anti-A and anti-B agglutinins was carried out with dried A and B blood group-specific substances of animal origin. The neutralization was successful, but there was a detectable loss of anti-Le^a activity at 37° C.

Owing to the graded strengths of reactions observed during the testing of Le(b+) cells, particularly of group A_1 with Mrs. Arm.'s serum, it was decided to perform all anti-X tests at 5° C., and to make the final reading after the slides had stood for one hour. This procedure proved satisfactory.

TABLE I.

Titres of Anti-X Serum (Mrs. Arm.) at 5°, 20° and 37° C.

Group O Test Cells.	Dilutions with Saline.				Temperature of Incubation.
	Undiluted.[1]	1 : 5.	1 : 10.	1 : 20.	
Le(a+)	4	3	2	1	5° C.
Le(a+)	4	2	1	0	20° C.
Le(a+)	3	1	0		37° C.
Le(b+)	4	1	0		5° C.
Le(b+)	4	0			20° C.
Le(b+)	0				

[1] Figures indicate degrees of agglutination.

Results and Discussion.

Neutralization Tests on Lewis Sera, Anti-Le^a, Anti-Le^b and Anti-X, with Various Selected Saliva Samples.

Brendemoen (1949), working with anti-Le^a serum, demonstrated Lewis group-specific substances in 41 (83·7%) of 49 saliva samples tested. The substances were found to be demonstrable in saliva from both Le(a+) and Le(a−) persons, but not in all samples tested. Grubb and Morgan (1949), working with human serum, saliva and ovarian cyst fluid, showed that both Le^a and Le^b substances exist, and that they possess the same general chemical, physical and serological characters as the A, B and H substances. In their tests they demonstrated that saliva samples at a dilution of 1:5 obtained from 30 Le(a+) persons, all neutralized anti-Le^a serum, while 42 (84%) of 50 saliva samples at the same dilution from Le(a−) persons also neutralized anti-Le^a serum. It was thought probable that all persons possessing the Le^a gene secrete Le^a substance, even if the presence of the Le^a gene is not demonstrable in the red cells of those in whom the genotype is Le^{a}Le^b.

The experiment summarized in Table II extends the observations published by the above-mentioned workers with anti-Le^a serum by including, in addition, neutralization tests with anti-Le^b serum and anti-X serum on four distinct saliva samples.

Neutralization tests on the three anti-sera were carried out with saliva samples boiled for twenty minutes immediately after collection, from persons of the following Lewis types: (i) group O, H non-secretor, Le(a+b−); (ii) group O, H secretor, Le(a−b+); (iii) group O, H secretor, Le(a−b−); (iv) group A_1, A non-secretor, Le(a−b−). The results of this experiment are shown in Table II.

The results summarized in Table II show the following points:

1. That anti-Le^a serum (four parts) was neutralized completely by saliva (one part) from an Le(a+) person, but incompletely by the same amount of saliva from an Le(b+) person. This person may, however, have been of Le^{a}Le^b genotype and not Le^{b}Le^b. When the latter saliva was increased from one part to two parts neutralization was complete—that is, one-half volume of saliva was needed. The latter proportion of saliva from group O, Le(a−b−), H substance secretor, and from a group A_1, Le(a−b−), A substance non-secretor, showed no neutralization when compared with a control test in which dilution was with the same volume of saline.

2. That anti-Le^b serum behaved as did anti-Le^a, except that the converse applied in relation to the saliva samples from the Le(a+) and Le(b+) subjects.

3. That anti-X serum (anti-Le^a + Le^b), in which the individual components were somewhat weaker than in the univalent sera, was neutralized when one part of saliva from either Le(a+) or Le(b+) subjects was added to four parts of serum.

It is obvious that the Le(a+) and Le(b+) subjects used in this experiment secrete both Le^a and Le^b substances, but in either case the homologous substance was secreted in slightly greater amount.

Further experiments were carried out, one volume of serum being used *plus* one volume of each of three Le(a−b−) saliva samples, again with saline controls. One group O sample of saliva from a person Le (a−b−), H substance secretor, neutralized anti-Le^a but not anti-Le^b; one sample from a person of group A_1 Le(a−b−), A substance secretor, neutralized anti-Le^b but not anti-Le^a; while a third saliva sample from a person of group O Le(a−b−), H substance non-secretor, did not neutralize either serum. The three subjects had been typed as unequivocally Le(a−b−). It is difficult to offer any concept which satisfactorily explains this lack of correlation between the presence of the Lewis substance in the saliva and its absence from the red cells of these three subjects. Possibly, if a larger series of such persons could be examined, the data obtained might reveal some underlying system.

The Lewis Blood Types in White Australians.

The Lewis sera described in this paper anti-Le^a, anti-Le^b and anti-X (anti-Le^a + Le^b) were used to determine the Lewis types in 500 white Australians. All tests with anti-Le^a serum were read after the slides had stood for

TABLE II.

Neutralization of Anti-Le^a, Anti-Le^b and Anti-X (Anti-Le^a+Le^b) Sera with Selected Saliva Samples.

Proportion of Saliva or Normal Saline.	Anti-Le^a Serum (Mrs. M.P.), 4 Parts. Results Read after 15 Minutes at 20° C.		Anti-Le^b Serum (Mrs. Mal.), 4 Parts. Results Read after 60 Minutes at 20° C.		Anti-X Serum (Mrs. Arm.), 4 Parts. Results Read after 60 Minutes at 5° C.	
	Le(a+) Group O Cells.	Le(b+) Group O Cells.	Le(a+) Group O Cells.	Le(b+) Group O Cells.	Le(a+) Group O Cells.	Le(b+) Group O Cells.
Le(a+) subject (H.B.), 1 part	—	—	—	2	—	—
Le(a+) subject (H.B.), 2 parts	—	—	—	—	—	—
Le(b+) subject (J.G.), 1 part	2[3]	—	—	—	—	—
Le(b+) subject (J.G.), 2 parts	—[4]	—	—	—	—	—
Le(a−b−)[1] subject (I.M.), 1 part	4	—	—	3	4	3
Le(a−b−) subject (I.M.), 2 parts	4	—	—	3	4	3
Le(a−b−)[2] subject (J.R.), 1 part	4	—	—	3	4	3
Le(a−b−) subject (J.R.), 2 parts	4	—	—	3	4	3
Normal saline, 1 part	4	—	—	3	4	3
Normal saline, 2 parts	4	—	—	3	4	3

[1] Group O subject, H secretor.

[2] Group A1 subject, A non-secretor (an exception).

[3] Figure indicate degrees of agglutination.

[4] "—" indicates no agglutination.

fifteen minutes at 20° C. in moist chambers; with anti-Le^b serum, after sixty minutes at 20° C.; with anti-X serum, after one hour at 5° C. The numbers of the blood samples tested in groups O, A_1, A_2, B and AB, together with the Lewis types detected, are shown in Table III.

Table III.

The Lewis Blood Types in White Australians.[1]

Blood Group.	Number of Blood Samples Tested.	Lewis Types.			
		Le(a+b−x+)	Le(a−b+x+)	Le(a+b+x+)	Le(a−b−x−)
O	212[2]	52 24·5%	148 69·8%	1 0·5%	11 5·2%
A1	150	48 32·0%	97 64·7%	0	5 3·3%
A2	50	11 22·0%	39 78·0%	0	0
B	53	12 22·6%	40 75·5%	0	1 1·9%
AB	35	9 25·7%	25 71·4%	0	1 2·9%
Totals	500	132 26·4%	349 69·8%	1[3] 0·2%	18 3·6%

[1] This series of 500 is additional to the 1123 Le^a tests reported by Simmons, Semple and Graydon (1950), of which type Le(a+) was found in 26·5%.

[2] An additional but earlier 100 group O samples tested with anti-Le^a and anti-X sera yielded only one Le(a−x−) sample. The anti-Le^b tests could not be performed at this time.

[3] A further group O person on our panel has subsequently given very definite Le(a+b+x+) reactions.

There is no significant difference in the percentages of the Lewis types found in the A B O blood groups. In the total of 500 samples tested, 132 (26·4%) were of type Le(a+b−X+), 349 (69·8%) were of type Le(a−b+X+), one (0·2%) was of type Le(a+b+X+), and 18 (3·6%) were of type Le(a−b−X−). The Le(a+b−X+) percentage of 26·4 is almost identical with the percentage of 26·5 found in 1123 white Australians in another survey. However, the percentage is higher than that found in England, Denmark and Norway, where it is just over 20 in each country. The Le(a−b+X+) percentage is almost identical with that found in Denmark. One sample in this series was found which was regarded as Le(a+b+X+), although the reaction obtained with anti-Le^a serum was weaker than that found in persons regarded as being of genotype Le^{a}Le^a. Subsequent tests on further members of our blood panel have yielded another example of type Le(a+b+X+) in a group O person. It would therefore appear that the dominance of Le^b over Le^a is not complete in all adult blood samples. The finding of these samples suggests that occasional blood samples do exist which justify their inclusion in this type. The percentage of Le(a−b−X−) was 3·6, compared with 6% found in Denmark. It would seem that another Lewis type antiserum (anti-Le^c) may yet be found. This suggestion is supported by the unequivocal examples of Le(a−b−X−) cells found, and by the difference in the ability of individuals of this type to secrete Le^a and Le^b substances, when compared with Le(a+) and Le(b+) persons who may secrete both substances in their saliva.

Summary.

1. The characteristics of three Lewis anti-sera anti-Le^a, anti-Le^b and anti-X have been described. An anti-Le^a serum reacting at 37° C. has remained fully active after being stored in the wet state for four years at 5° C. An anti-Le^b serum, when detected at delivery, exhibited all the characters of the anti-Le^b serum as described by Andresen. Seven days later this serum no longer showed the complete epitasy effect, and was capable of reacting with all Le(a−) cells at 37° C. irrespective of their A B O blood group. An anti-X serum detected after a transfusion reaction was found to increase in titre after delivery of a normal baby. From the observations made it is suggested that the anti-X serum investigated by us and possibly that described by Andresen and Jordal are actually examples of polyvalent anti-Le^a + Le^b sera.

2. Neutralization experiments carried out with anti-Le^a, anti-Le^b and anti-X sera and four different saliva samples have shown that the Lewis activity of the three sera can be neutralized with saliva from either Le(a+) or Le(b+) persons. Such persons may secrete both Le^a and Le^b substances, but the homologous substance is secreted in greater amount. Under the conditions of the original experiment, samples of saliva from persons of type Le(a−b−), H secretor, and Le(a−b−), A non-secretor, were both without neutralization effect. However, when a proportionately greater volume of saliva was added to the serum, the saliva of the first type showed the presence of some Le^b substance but no Le^a substance, while in that of the second type there appeared to be no detectable Le^a or Le^b substance. Another neutralization test carried out at a later date with saliva from a group A_1 Le(a−b−), A substance secretor, showed the presence of some Le^a substance but no Le^b substance.

3. The Lewis blood types have been determined in 500 white Australians. There was no significant difference in the percentages of the Lewis types found in the A_1, A_2, B, O and AB blood groups. In the total of 500 samples tested, 132 (26·4%) were of type Le(a+b−), 349 (69·8%) were of type Le(a−b+), one (0·2%) was of type Le(a+b+), and 18 (3·6%) were of type Le(a−b−).

4. The possibility is briefly discussed of the existence, as already suggested by others, of another Lewis type antiserum, anti-Le^c.

Acknowledgements.

We are grateful to Mr. N. M. Semple, B.Sc., for his many acts of assistance in this work, and to Miss Frieda Lottkowitz for the collection of all blood samples, and the keeping of records. Finally, we express our appreciation and thanks to the three women patients for their generous blood donations and cooperation in this investigation.

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CARCINOMA OF THE AMPULLA OF VATER.

By CHARLES OSBORN, F.R.C.S., F.R.A.C.S.,
From The Surgical Service, Saint Vincent's Hospital, Melbourne.

FOR its size there is probably no tumour more lethal than carcinoma involving the ampulla of Vater. Such a provocative statement will at once be challenged, in view of the fact, which most clinicians know, that such a tumour infiltrates slowly and metastasizes late (Gray and Sharp, 1943). It is not the degree of malignancy that determines the lethal quality of this tumour, but merely its site, because a minute tumour here can obstruct the bile duct and in the short space of a few months cause death from liver failure in a state of deep jaundice.

Tumours at the ampullary site may arise in the termination of the common or the pancreatic duct, from the ampulla of Vater itself, or from the intestinal mucosa of the papilla, and in many cases it will be impossble to determine the exact site of origin (Hunt, 1941).

Ampullary tumours, although not rare, are certainly far from commonplace, and it is difficult in many cases to distinguish them pre-operatively from carcinoma of the pancreatic head; but it is important that such distinction should be made, because although an experienced surgeon may be deterred from radical resection for pancreatic carcinoma because of the somewhat unsatisfactory late results (Cattell and Pyrtek, 1949), the very character of the ampullary growth, the small size and the early declaration of its presence, make this a favourable tumour for radical resection with reasonable prospects of a permanent cure.

Until the work of Whipple, Parsons and Mullins, who in 1935 published a technique of radical pancreaticoduodenal resection, malignant tumours of the ampullary region were usually treated by local resection with some plastic procedure to the common bile duct and cholecystenterostomy, or simply by palliative short circuit of the biliary apparatus. Results in the main were far from satisfactory, and few patients survived for more than a few years. This was not surprising, because the principles underlying the surgical treatment of malignant disease were inadequately carried out by such local excision, and the immediate operative mortality rate was high, on account of the deep jaundice and our lack of knowledge at that time of vitamin *K*, together with what we now know to be an inadequate pre-operative preparation of the patients with disturbed liver function.

As in many other surgical fields, recent additions to our knowledge relating to pre-operative preparation, anti-shock measures, anæsthesia and the newer antibiotics have enabled the surgeon to undertake much more radical procedures than were previously contemplated, and these advances have particular application to the tumour under discussion; here adequate resection entails wide excision in a particularly vascular field and necessitates multiple anastomoses to reconstitute functional pathways.

Baggenstoss in 1938, and more recently Cattell and Pyrtek in 1950, have drawn attention to the occasional presence of benign tumours of the papilliform or adenoid type arising from the ampulla. The significant conclusion of these authors is to emphasize the malignant tendencies of such tumours.

As the average life expectancy of patients with carcinoma of the pancreas and ampullary region is only about six months (Whipple, 1949), and as it is a fact that the operative mortality for radical resection has fallen considerably in recent years (to the vicinity of 10% or less), the modern surgical view may be stated to be that radical surgical intervention should be a worthwhile procedure in selected cases.

Cattell (Cattell and Pyrtek, 1949), after an extensive personal experience, is far from enthusiastic in his support for radical resection in cases of cancer of the pancreas, but records twelve patients suffering from carcinoma of the ampullary region followed for five years or more, three of whom show no evidence of recurrence.

Several authors have reported small series of patients who have been submitted to radical pancreatico-duodenal resection for ampullary carcinoma with satisfactory long-term results, and so I take this opportunity of recording the clinical and operative details of two patients upon whom I operated in 1947 and 1948 respectively, and who are at present alive and in perfect health three years and two years later.

Reports of Cases.

CASE I.—E.M., aged forty-nine years, a male patient, was admitted to hospital in August, 1947. He said that twelve weeks previously he had suffered from an attack of "ulcer pain" for one week, which simulated the pain he had experienced several years previously when the condition had been diagnosed and confirmed radiologically as due to a duodenal ulcer. Then six weeks later he noticed that he was losing weight, was not enjoying his food and was suffering from "indigestion", but had no "ulcer pain". Shortly after this the urine became noticeably darker and his skin was beginning to itch. These symptoms continued, and ten days later jaundice was noted and the stools became pale in colour. No real pain was experienced, and after unsuccessful treatment for the jaundice and intolerable itching he was referred to hospital for treatment. His appetite was very poor, and 14 pounds of weight had been lost; his bowels acted daily, but were sluggish, and abundant laxatives were taken. Nausea had become a prominent symptom during the two weeks prior to his admission to hospital, and occasional vomiting afforded some relief.

Physical examination revealed the patient to be a thin man, deeply jaundiced, with many scratch marks on the trunk and limbs in all accessible places. An enlarged gall-bladder could be faintly outlined by the palpating hand, but no enlargement of the liver was detected. Irregular fever was present during the first two weeks in hospital. Laboratory findings at this time were as follows. The blood urea content was 42 milligrammes *per centum*, the plasma protein content was 6·9 grammes *per centum* and the albumin-globulin ratio was 1·8:1·0. The serum bilirubin content was 14 milligrammes per 100 millilitres, the icteric index was 91, the alkaline phosphatase was 30 units. The cephalin flocculation test produced a negative result, and the Van den Bergh test produced a direct positive response. The prothrombin index was 80, the hæmoglobin value was 80%, examination of the fæces for occult blood gave negative results, and the response to the hydatid complement fixation test was negative. A diagnosis of obstructive jaundice the result of neoplasm was made, and a preoperative régime was instituted of vitamin *K* injections, glucose drinks, a protein-rich and fat-free diet, together with orange juice and vitamin *B* complex. A blood transfusion was given on the day of operation.

The details of the first operation are as follows. Under spinal anæsthesia, the patient's abdomen being lax, a mass was shown elevating the skin under the right costal margin, about two inches above and lateral to the umbilicus. A transverse incision about six inches long was made under the right costal margin and the abdomen was opened. A large distended gall-bladder projected into the wound. On palpation no stones were felt in the gall-bladder, the cystic or the common duct, but at the ampulla of Vater a mass the size of a pea could be felt. A cholecystostomy was performed, the tube emerging at the most lateral part of the incision. The abdomen was closed in layers, no other drainage being used.

Six weeks elapsed before the second operation, during which time bile drained freely through the tube and the jaundice almost disappeared. Four ounces of bile were introduced into the stomach by Rehfuss tube after each meal. The laboratory findings now were as follows. The blood urea content was 35 milligrammes *per centum*, the plasma chloride content (as sodium chloride) was 538 milligrammes *per centum*, the plasma protein content was 6·9 grammes *per centum*, the serum bilirubin content was 0·6 milligramme *per centum* and the icteric index was 24.

The details of the second operation are as follows. Controlled intratracheal cyclopropane anæsthesia was used with curare. The abdomen was opened by a right paramedian incision. A few adhesions were encountered and divided in the upper part of the wound. Considerably more were revealed in the peritoneal cavity to the right of the area exposed by wide retraction. The tumour in the ampulla of Vater about the size of a pea was again palpated, but with

some difficulty. The greater curvature of the stomach was freed by division of the gastro-colic omentum between clamps and ligatures. The lesser omentum was then divided and the stomach was transected through the antrum.

A curious abnormality detected was the origin of the middle colic artery from the gastro-duodenal branch of the hepatic artery, which precluded division of the gastro-duodenal artery. Division and ligation of numerous branches of the gastro-duodenal artery were necessary at a later stage of the operation.

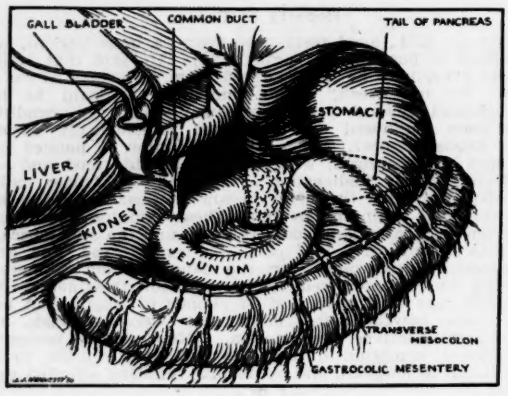

FIGURE I.

Diagrammatic representation showing the anastomosis of the pancreas, common duct and stomach to the jejunum after resection; cholecystostomy is also illustrated.

The duodenum was mobilized after division of peritoneum on the right lateral aspect. The common bile duct was dissected free to the upper border of the pancreas and then divided between clamps, as was the third part of the duodenum through the horizontal part. The head of the pancreas was then divided between clamps and the isolated mass of duodenum, pancreas and common duct was removed in one piece. The pancreatic duct was revealed and intubated with a rubber tube of small diameter. The distal end of the cut duodenum was invaginated. The jejunum about 18 inches from its origin was divided between clamps. The distal portion was brought through the meso-colon and the open end was anastomosed to the cut end of the head of the pancreas with the tube still in the pancreatic duct. The choledochus was grossly dilated and was anastomosed end to side with the jejunum. The proximal cut end of the stomach was then partly sutured and the remainder anastomosed end to side to the jejunum. The proximal end of the previously divided jejunum was anastomosed end to side into the jejunum lower down. Inspection of all anastomoses was finally carried out and all seemed satisfactory.

An area of oozing in the region of the divided head of the pancreas, which could not be controlled by ligatures, was tightly packed with gauze tape. Drainage was provided by corrugated rubber, which was placed into the choledochal area alongside the gauze tape. The whole operation area was lightly dusted with a fine spray of penicillin and sulphanilamide powder and the abdominal wall was closed in layers.

On October 17, 1947, the pathologist, Dr. Andrew Brenan, reported as follows on the tissue removed:

> Masses of glands of various sizes spread through the wall and in some areas infiltrate deeply through the muscle. Many glands are well formed and are lined by tall columnar cells or by cubical epithelium. Some are irregular and some show lining cell hyperplasia. Where this hyperplasia is marked some nuclei are larger and some show hyperchromatism: a few show chromolysis and very few mitoses. There is marked intrapapillary formation in some dilated glands.
>
> The tumour is an adeno carcinoma.

During post-operative convalescence vomiting was troublesome and necessitated gastric aspiration for several days; it was assumed that œdema at the gastro-jejunal anastomosis accounted for the retention of stomach contents. However, after a few days the stomach fluid balance appeared favourable and the indwelling gastric tube was withdrawn.

For nine days there was free discharge of bile-stained fluid from the abdominal wound along the drainage tube placed in the neighbourhood of the common duct, jejunal and pancreatic anastomoses. Examination of this fluid revealed the presence, not only of bile, but also of trypsin and diastase. Leakage was occurring at the anastomotic sites; but as suitable drainage and an alterative route for the bile through the cholecystostomy tube had been provided, no fear was occasioned for satisfactory closure, which took place on the tenth post-operative day.

Some little time elapsed before this patient could tolerate a liberal diet. Thirty days after his operation he was discharged from hospital taking a full diet and feeling very well, and now three years later he remains in excellent health, pursues laborious work and has no bowel disturbance. He has regained his normal weight and looks well. A cholangiogram revealed adequate patency of the common duct-jejunal anastomosis.

The surgical approach to this case consisted of two stages—not the orthodox two-stage operation, in which the first stage consists of exploration and then internal anastomosis of the extrahepatic biliary tract to the alimentary canal by either a cholecystenterostomy or choledochoenterostomy, as from previous experience in cases of carcinoma of the head of the pancreas this procedure impedes the subsequent exposure of the second operation. The first stage was purely one of exploration and confirmation of diagnosis, followed by a simple cholecystostomy established well out toward the right extremity of the transverse incision made in the right hypochondrium. The usual signs of ampullary tumour were revealed—namely, dilated extrahepatic bile ducts and distended gall-bladder—and through the duodenal wall could be felt a firm globular tumour about the size of a pea at the site of the ampulla, whilst the head of the pancreas appeared normal.

Eight weeks of deepening jaundice preceded this operation, and it was believed that there would be less hazard when the radical operation was carried out if a preliminary decompression was performed, the liver function thus being enabled to return to normal, and if the patient was gaining weight with a restored digestion. The risk of hæmorrhage was never a cause for anxiety because of vitamin *K* therapy, and after several weeks of biliary drainage together with reintroduction of bile through a Ryle tube and appropriate protein-rich diet, it could not be denied that a much fitter patient presented for the second operation.

During post-operative convalescence the presence of a safety-valve cholecystostomy proved useful, and this functioned during the early stages freely. This was to be expected, and was designed to take care of œdema at the common duct-jejunal anastomosis which most surely occurs during this period.

A knowledge of the variable vascular anatomy (Ziegler, 1942) proved valuable in this case, wherein the middle colic artery arose from the gastro-duodenal artery and coursed along in front of the pancreatic head and the horizontal portion of the third part of the duodenum. Early ligature of the gastro-duodenal artery, a recognized part of the routine, would in this case have endangered the blood supply of the transverse colon and thus necessitated additional shock-producing surgery at the end of an already long operation. This vascular anomaly provided an irritating obstacle throughout the operative procedure and effectively prevented the easy control of the arterial supply to the region.

Division of the duodenum was made at the commencement of the ascending limb of the third part, a blind end thus being left which required subsequent anastomosis. No particular advantage was gained from this procedure (nor has it been repeated) designed to avoid the somewhat difficult and dangerous dissection entailed in freeing the duodeno-jejunal flexure from beneath the superior mesenteric vessels.

CASE II.—J.T., a male subject, aged sixty-two years, was admitted to hospital in October, 1948, complaining of jaundice of two weeks' duration. He had had no previous

illnesses. He was a rubber worker and did not handle chemicals. He had mined gold for ten months, but did not use trinitrotoluene for blasting. He had no family history of disease. His present illness dated from six months previously. Whilst away mining he first felt "off colour" and noticed indigestion and a slight itch. Some loss of weight was noted and the urine became dark. Two weeks prior to his admission to hospital he vomited nine times in one day (he had vomited on only two occasions since). He then began to suffer from severe itching and noticed that the urine was very dark. His motions became white and were firm and offensive. He had no history of melæna or abdominal pain. The patient volunteered the information that the jaundice was now not so deep as it had been.

On physical examination of the patient his abdomen was lax and moved well with respiration. The liver was palpably enlarged three fingers' breadth below the costal margin, liver dulness extending up to the seventh costal cartilage. The liver, visibly moving on deep inspiration, was large and smooth with a rounded lower edge. The gall-bladder was palpable and greatly enlarged. No other abdominal mass or viscus was palpable. There was deep jaundice, and scratch marks on the trunk and limbs were noticeable.

The laboratory findings were as follows. The blood urea content was 50 milligrammes *per centum*, the plasma protein content was 5·3 grammes *per centum* and the serum alkaline phosphatase content was 30 units. The hæmoglobin value was 94%, the cephalin flocculation test produced a negative result, the prothrombin time was 90%, and the serum bilirubin content was 16·0 milligrammes *per centum*. Examination of the fæces for occult blood gave negative results.

A clinical diagnosis of obstructive jaundice due to a tumour about the ampulla of Vater was made, and operative treatment was advised and carried out after suitable preparation.

The abdomen was opened by an upper transverse incision and the gall-bladder and bile ducts were found to be grossly dilated. The liver was swollen and green, but there was no evidence of metastatic carcinoma therein. A small hard nodule about the size of an orange "pip" was felt at the site of the ampulla of Vater, and there appeared to be some thickening in the neighbouring pancreatic tissue.

Radical pancreatico-duodenectomy was decided upon. The gall-bladder and ducts were emptied and the common duct was divided between clamps, after the duodenum had been mobilized by division of the peritoneum along its right lateral border. The stomach was clamped across the pyloric antrum by de Martel's clamps and divided; the gastro-duodenal artery being then exposed was ligated and divided. Complete mobilization of the duodenum was carried out until the vicinity of the duodeno-jejunal flexure was reached, when the gut was cut across and the inferior pancreatico-duodenal artery was ligated. Separation of the head of the pancreas from its posterior relations, by following the plane of the anterior surface of the portal vein, made possible division through the neck of the gland, and the mass consisting of the pyloric antrum, the duodenum, the head of the pancreas and the terminal portion of the common bile duct was removed *en bloc*. A further six inches of jejunum were removed to make adequate mobilization possible, and the cut end was brought through the mesocolon and sutured over the exposed cut surface of the pancreas, a single layer of interrupted silk sutures being used. The common bile duct was then anastomosed end to side to the jejunum several inches below the pancreatic anastomosis, again with a single layer of interrupted silk sutures. A gap of about fourteen inches intervened between this and the next anastomosis between the cut end of the stomach and the side wall of the jejunum, as in Polyá gastrojejunostomy. The jejunum was anchored to the posterior abdominal wall in the neighbourhood of the hilum of the liver to prevent any undue tension from developing near the choledocho-jejunal anastomosis, and the mesocolon was sutured to the traversing jejunal limb.

After a tube had been sutured into the gall-bladder and a corrugated rubber drain had been placed in position, the abdomen was closed in layers.

The pathological report on the specimen (Dr. A. Brenan) was as follows:

> A small hard nodule not much larger than a grain of wheat right at the ampulla of Vater. The tumour consists of a mass of glands many of which are lined by tall columnar cells. Some are dilated and irregular and some show lining cell hyperplasia. Glands can also be seen in the muscular layer. Some other glands are small and well defined. I would regard it as an adeno carcinoma.

The period following operation was uneventful. Oral feeding was commenced on the fourth post-operative day and the cholecystostomy tube was withdrawn on the twentieth day after a cholangiogram had been prepared. The serum bilirubin content was 3·0 milligrammes *per centum* on the twenty-eighth day and reached a normal level (0·2 milligramme *per centum*) on the forty-seventh post-operative day.

The subsequent history of this patient has been satisfactory. Except for occasional slight biliary discharge from the wound at odd times during the first few months he has led a normal life and has resumed his occupation. There are no bowel disturbances, he has regained his weight, and after two years he feels and looks well.

In spite of the relatively short history in this case there was evidence of damage to the liver, which at the operation was "swollen and green". A one-stage operation was determined upon in this case because of the relatively short history, and because of the fact that the patient responded well whilst in hospital awaiting operation.

Again the safety-valve cholecystostomy was employed; but this time it was made at the end of the operative procedure and not as a preliminary operation as in Case I. This proved useful during convalescence and discharged bile freely during the early stages, whilst only a small amount of bile was discharged along the abdominal drain.

The Diagnosis of Carcinoma of the Ampullary Region.

The first essential is to determine that the patient under consideration is the subject of obstructive jaundice. This sometimes proves difficult, and a little delay is often necessary whilst liver function tests are carried out and perhaps repeated. Confusion can arise in a variety of ways. (i) If jaundice has been present for some time and is very deep, then liver damage will have occurred to a degree severe enough to produce laboratory findings consistent with hepatitis. (ii) Fever may accompany the jaundice of obstruction—even that due to carcinoma—whilst enlargement of the liver is present in both hepatitis and advanced obstructive jaundice. (iii) Although the enlarged, tense gall-bladder is diagnostic of obstruction, it may, of course, not be present if there is cholelithiasis. This occurred in one patient suffering from carcinoma of the head of the pancreas with obstructive jaundice and associated gall-stones.

Once the conclusion has been reached that the jaundice is obstructive, a decision upon the site or cause of the obstruction, although less important, becomes an interesting clinical exercise. The commoner causes of primary obstructive jaundice are common duct calculi, carcinoma of the head of the pancreas and ampullary or periampullary carcinoma, whilst more rarely seen are carcinoma of the common duct, benign tumours of the ampulla, fibrosis of the papilla and chronic pancreatitis.

In the usual order of sequence the symptoms and signs are as follows.

1. Loss of weight and feeling "off colour" often precede the more characteristic symptoms by several weeks.

2. Itchiness of the skin usually precedes the jaundice by one to three weeks, and during this time the patient usually notices that the urine is becoming progressively darker.

3. Jaundice is steadily progressive in most cases; but it should be remembered that with ampullary lesions remissions of the intensity of the jaundice may be noted. These are apparently due to ulceration of the growth which permits escape of bile and thus lessens the intraductal tension. This fluctuation of the jaundice will cause confusion and simulates a stone in the common duct; but with ampullary lesions the gall-bladder is commonly distended (Courvoisier's law). Jaundice occurs early in ampullary lesions, because a very small tumour at this site will soon interfere with the discharge of bile into the duodenum. It is common, therefore, to see a severe degree of jaundice in a patient who, apart from the jaundice and some loss of weight, still looks fairly well, in contradistinction to a patient whose growth is primarily in the

pancreas, and who with a similar degree of jaundice will look emaciated and ill. The Van den Bergh reaction is directly positive, and the serum bilirubin level will be raised to about 18 milligrammes per 100 millilitres in the first few weeks, rising to higher levels as the jaundice deepens further. Bile disappears from the stools, which become clay-coloured; but it is rare for the stool to be noticeably bulky and greasy.

4. Pain usually occurs. "Painless jaundice" has become axiomatic for carcinoma of the pancreas or ampulla. However, modern authors are agreed that such a statement is incorrect, and in fact pain is an initial symptom in about half the cases and sooner or later occurs in almost all cases. Pain is usually much more pronounced in carcinoma of the pancreas. In ampullary carcinoma the pain rarely becomes severe, and an early complaint of indigestion frequently precedes the jaundice. This constitutes a difference from the pain due to pancreatic carcinoma or gall-stones.

5. Vomiting occurred during the illness of both the patients whose histories are recorded herein; but it is not a prominent symptom, whereas nausea and loss of appetite are common features in all cases.

6. Hæmorrhage from an ampullary growth sufficient to provide a positive response to the benzidine test on the fæces is common, and is regarded as a valuable diagnostic sign. It was not present in either of the cases quoted here, nor was this surprising when the growths were examined, for in each instance the growth was small and no surface ulceration could be detected. Occasionally melæna may occur.

7. Enlargement of the gall-bladder is customarily regarded as diagnostic of non-calculous obstruction; but the absence of the sign is by no means infrequent (Aird, 1949) and is difficult to explain.

8. Enlargement of the liver is usually demonstrable when the jaundice has been present for a few weeks.

9. Ampullary carcinoma is twice as common in the male sex (Siler and Zinninger, 1948), the main incidence being in the fifth and sixth decades.

From the foregoing it will be seen that the diagnosis of ampullary carcinoma can be made with reasonable certainty only in the case of a middle-aged man who has deep jaundice with only mild pain and slight loss of weight, and who presents with an enlarged gall-bladder and occult blood in the stools.

X-ray studies have been of little diagnostic value (Hunt, 1941); occasionally filling defects have been noticed in the second part of the duodenum in the vicinity of the papilla of Vater.

At operation, it may be difficult to identify the tumour, and it will certainly be difficult in many cases to be sure of the exact pathology.

When the tumour is very small it may be difficult to palpate through the duodenal wall—it may be difficult in any case to distinguish from a stone obstructing the ampulla. When doubt exists it will be necessary to inspect the site of the papilla after incision of the duodenum, and thus to exclude calculi; but in the case of benign tumour doubt cannot be resolved until the pathologist's report is available—the immediate frozen section finding is untrustworthy.

Remarks on the Technique of Pancreatico-Duodenectomy.

An extensive literature exists on the technical problems relating to the operation of pancreatico-duodenectomy; but gradually over the fifteen years since Whipple first recorded his successful case an agreement has been reached and the principal steps have been more or less standardized.

Operability.

Ampullary tumours are usually favourable for resection, because diagnosis is made reasonably early, metastases are late in appearing, and local spread has rarely invaded neighbouring structures (Brunschwig, 1942).

Staged Operations.

Whereas earlier authors strongly recommended a two-stage operation, the first stage consisting of anastomosis of the gall-bladder or common bile duct to the duodenum, there has been a tendency in recent years (excepting by Cattell) to complete the operation in one stage.

The advantages claimed for primary cholecystostomy as used in the first case here recorded are that a severely damaged liver and poor nutritional state may be restored to nearly normal without any interference with the site of the subsequent operation of resection. It is true that Cattell claims that cholecyst-jejunostomy as the first stage does not hamper the second operation, but my own experience does not confirm this view. Particularly is this so when the common duct and not the gall-bladder is used in the anastomosis, in which case the jejunal loop is closely approximated to the anterior surface of the duodenum.

Cholecystostomy entails the necessity of feeding back the bile to the patient through a Levine tube; this is a somewhat unpleasant procedure; but it is satisfactory, and if the cholecystostomy is made far out in the right hypochondrium no unpleasant adhesions will mar the future operation site.

In cases in which liver damage is nominal and nutrition is satisfactory, the operation may be carried out in one stage, but again terminating with a cholecystostomy for reasons already cited.

The Defunctioned Jejunal Limb.

By the defunctioned jejunal limb is meant a long limb of jejunum (14 to 18 inches) through which no stomach contents pass. It is simply constructed, no entero-anastomosis is required, it can be manipulated easily, and most important of all it is sterile and permanently free of stomach contents; thus the risk of infection to the liver or pancreas is minimized (Figure I). The order of the anastomosis is similar to that described by Child, 1944, wherein the cut surface of the pancreas is anastomosed to the proximal open end of the jejunum by interrupted silk sutures; next the divided common duct is united end to side about four inches further along the limb; and then finally the cut end of the stomach, partially sewn as in the Hofmeister modification of the Polya procedure, is united to the jejunum at least 14 inches distal to the previous anastomosis with the common duct. This length is considered sufficient to prevent any regurgitation proximally.

For convenience the defunctioned limb is taken through the mesocolon, which is subsequently snugly sutured to it. It will be found that the jejunum lies in a gentle S curve above the mesocolon.

In most other methods of hook-up the stomach contents will pass over the sites of anastomosis of the common duct and pancreas, and this applies even when a jejunal loop is used with an enteroanastomosis, as X-ray studies reveal that such enteroanastomosis is not a certain insurance that the loop is defunctioned. Furthermore, an extra anastomosis is entailed which can be avoided by using the single limb.

Anastomosis.

Anastomosis to the cut surface of the pancreas requires delicate and somewhat difficult suturing, as the capsule of the gland is fragile and ill-defined, whilst sutures hold poorly in the friable pancreatic tissue. It seems needless to intubate the pancreatic duct, although this was done in one of the cases here recorded. It is probable that the duct remains patent without either intubation or suturing.

Most authors are agreed that anastomosis of the common duct to the jejunum is much safer than any form of anastomosis using the gall-bladder, because of the liability of a blowout if the common duct is ligated. This anastomosis is the most difficult part of the operation, because not only is the common duct of tissue-paper thinness in its dilated state, but it may be uncomfortably short and thus prove difficult to manipulate smoothly. A single layer of through-and-through sutures at intervals

of about two millimetres seems to be adequate and all that I can place in this delicate structure without tearing it. Some leakage will almost certainly take place, but this is cared for by the corrugated drain placed down to the site.

Summary.

Two cases of carcinoma of the ampulla of Vater are presented, the patients having survived the operation of pancreatico-duodenectomy for three and two years respectively.

The diagnosis is discussed, and it is indicated that a diagnosis may be achieved at an early stage in the disease and that radical treatment may be undertaken with a reasonable chance of permanent success.

The principles of the operation are discussed and some of the technical details are elaborated.

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THE LIMITATIONS OF A PURE PSYCHOGENESIS.[1]

By BARRY MULVANY,

Lecturer in Psychiatry, Saint Vincent's Hospital Clinical School, Melbourne.

To acknowledge the limitations of anything does not imply destructive criticism, but tends to emphasize its rightful uses. The history of medicine is filled with hypotheses and procedures which by subsequent exaggeration have been wrongfully relegated to total discredit, only to be revived again in another age. Much time could be spent on the semantics of the term "psychogenesis". For those who are interested it is discussed at some length by Professor John Reid of Stanford University (1948). He suggests that the term psychogenesis implies that "a certain mental act perhaps best called simply an act of interpretation, *temporally preceded* and—as we suppose—*causally mediated*, the emergence of the symptoms in question" Reid amplifies this at some length. He might have adhered to his simple dictionary definition, "as originating in the mind", which he thought too simple. Incidentally he quotes Bernfeld to justify the extension of his definition to the role of the unconscious. Conversely, when psychotherapy succeeds in bringing the earlier "act of interpretation" into consciousness, discharging its affective content with perhaps a formal reinterpretation, so that the relevant symptoms disappear, then the causal role of the psychogenesis is beyond doubt.

The use of the word "pure" in this context is owed to Freud, for in his monograph on "The Question of Lay Analysis", having outlined his dynamic psychological theory, he makes his hypothetical listener exclaim with the inevitable pun: "Everything you have told me so far has been pure psychology. It has often sounded strange, or a bit thin or obscure, but it has been pure psychology in both senses." Yet in this paper the words "primary", "basic" or "specific" might be substituted.

The limitations of a pure psychogenesis must be considered from the ætiological and from the therapeutic aspects. In what proportion of psychiatric disorders does a pure or primary psychogenesis form the basis of the ætiology? And of this group, in what proportion does the recognition of this basic psychogenesis lead to therapeutic success? It is not the purpose of this paper to belittle the importance of psychological factors in psychiatry. In fact, its purpose is to extend the recognition of their influence to the neighbouring fields of medicine and surgery. The psychotherapeutic approach must remain the cornerstone of psychiatry even when structural pathology forms the basis of the disorder.

From time to time one sees psychiatric disorders elucidated in terms of a relevant psychogenesis and responding dramatically to specific psychotherapeutic procedures. Among the most dramatic are the psychoneuroses of war, in which the recall of the repressed traumatic incident, with its accompanying release of emotional tension, abolishes the symptoms and produces a contented and sociable individual. Less dramatic but no less impressive are the results in some cases of longer and more involved analytical procedures. Unfortunately, when one scans the whole field of psychiatry, observing what is known of ætiologies, and seeking some degree of certainty in therapeutic results, the prospect is anything but clear.

One of the commonest misconceptions about psychiatry is ignorance of its scope. It comes as a surprise to many people to find that the majority of disorders listed in the official classifications, either British or American, are organic disorders with prominent toxic, infective, or degenerative pathological basis. Congenital mental defectiveness, epilepsy and disorders of the vascular degenerative group, form a large proportion of the field of psychiatry. In our hospitals there are large numbers of such disorders, the greater concentration being found in State mental hospitals. Professor Aubrey Lewis emphasized the importance of the senile and aged group of patients, in an address to the Royal Medico-Psychological Association in 1945. This group provides the greatest number of first admissions to mental hospitals in Great Britain, while the figures for New York State are twice that of any other psychotic group. It is usually assumed that the psychiatrist deals almost exclusively with psychogenic disorders, and that the main metamorphosis in his development has occurred in a department of psychology.

When the large number of primary organic disorders are excluded from the field of psychiatry, there remain the all-important groups of the psychoneuroses, and the major psychoses of the schizophrenic and manic-depressive groups. The ætiology of these major psychoses is unknown, constitutional factors having a wider acceptance among psychiatrists than purely psychogenic factors. Yet it is probable that the majority of psychiatrists adhere to total conceptions involving an interaction of psychogenic and constitutional aspects. It is sometimes perplexing to listen to an elucidation of the ætiology of a psychosis in terms of a pure psychogenesis, and then to trace the subsequent history of the patient through electroconvulsive therapy and insulin coma, even to leucotomy. It is even more baffling to hear the same psychodynamic factors invoked in widely different psychotic reactions and in psychopathy. Repetition may weary the legal profession to the point of disbelief and an insistence on hanging.

[1] Read at a meeting of the Australasian Association of Psychiatrists, October, 1950, at Melbourne.

In the recent Linacre lecture, F. M. R. Walshe made a devastating attack upon medical psychology. He did not use the term "psychiatry", and one wonders whether he realized that modern psychiatry has inherited a multitude of disorders neglected by general medicine. Of the bed state in this country, Great Britain and America, 50% are psychiatric beds, for psychiatry, as Strauss has said, is "the other half of medicine". No branch of medicine defends such a wide and varied front, representing disorders of widely differing ætiologies, presenting the largest share of human suffering with hideous social consequences. Modern physicians and surgeons have carved out narrow impersonal specialties for themselves, tearing the fabric of the Hippocratic tradition and preparing the way for the piecemeal departmental management of the patient. From the confines of a narrow stylish specialty in which therapy is often left to the surgeon, Walshe states that psychological medicine "has become a chaos of conflicting allegorical systems, of pseudomysticism and of 'hit or miss' mutilations of the brain that have no ascertained or scientific basis". Yet much of his criticism is well reasoned and just, as is his argument that medical psychology belongs to the historical and not the natural sciences. He states that "the medical psychologist's problem is a concrete historical one and does not lead to the formulation of universals, for there is no blueprint, no master key, to the springs of human thoughts and actions, applicable to all persons at all times and in all circumstances, and no natural laws for the human mind, *as natural laws are generally conceived*".

It is beyond the scope of this paper to cover fully the arguments in favour of a constitutional or somatic basis for the major psychoses, either schizophrenic or manic-depressive. Much has been written of their genetic aspects and their incidence in twins. Some psychiatrists discount a psychogenesis entirely, emphasizing the response to physical treatment. There are many difficulties in the way of accepting a primary psychogenesis for these disorders. One of the most cogent arguments that there is a somatic determinant, is that the Kraepelinian grouping exists at all. It is now recognized that mixed psychotic reactions with both manic-depressive and schizophrenic features do occur, but the classical Kraepelinian reactions stand out like primary colours in the psychotic spectrum. With a pure psychogenesis one might expect a multiplicity of syndromes with no high correlation between certain symptoms. That such correlation exists has been established by T. V. Moore as quoted by Henderson and Gillespie. Moore confirmed Kraepelin's differentiation of psychotic syndromes by making a statistical analysis of a large number of psychotic symptoms and then establishing the degree of correlation between them. A high correlation of symptoms corresponding to Kraepelinian entities was found. Hence the possibility that a somatic determinant shapes these entities becomes more certain.

The statistical incidence of various disorders in varying races, cultures and social strata is of considerable importance. Thus the incidence of schizophrenia and of manic-depressive psychosis in Kenya Africans compared to their incidence in American Negroes, has been investigated by Carothers (1947). The incidence is the same in both groups, as is that of epilepsy, despite gross differences in family, tribal and social organization. The difference between the groups in infantile and adolescent sexual attitudes and customs is immense. Before we leave the statistical grouping of psychiatric syndromes, mention will be made of the high incidence of abnormal electroencephalograms among psychopaths and mixed psychoneurotics, as demonstrated in the work of Hill and Williams. The abnormality is non-specific. Williams summarized the position by stating that "an abnormal E.E.G. in an otherwise normal subject is strong evidence of an inborn constitutional abnormality involving the central nervous system. This abnormality appears to be non-specific, and may manifest itself in the subject or his offspring as a behaviour disturbance which may be psychoneurotic, psychopathic, psychotic or epileptic in type". It is the belief of D. K. Henderson, after lifelong study, that psychopathy is a constitutional state. The demarcation of the psychotic, psychopathic and psychoneurotic groups from the bulk of gaol populations in the studies of East and others is also a matter of interest. Holt analysed the histories of 5000 persons committed to the Massachusetts gaols after examination by the Department of Mental Diseases, and found that the diagnosis of psychopathic personality was made in 15·5%. The attempt to link the groups with the ordinary offender and in turn with the so-called normal through the medium of a basic psychogenesis and this "formulate universals" is what Walshe and others find incomprehensible.

One of the great arguments for the primary psychogenic causation of the major psychoses is the absence of any discoverable structural pathological basis despite intensive work over many years. The apparently meaningful and sometimes well-patterned behaviour of the schizophrenic tends to reinforce the concept of a psychogenesis, as do the occasional reported cases of remission following analytical treatment. Yet remissions in schizophrenia have been noted under varying conditions for many years. The absence of gross pathological change does not exclude a multitude of possibilities such as involvement of intracellular enzymic systems or humoral abnormality.

In general medicine an analogy is provided by rheumatoid arthritis, which eluded the pathologists for many years. The discovery by Hench and Kendall that cortisone exerted a dramatic effect, objectively and subjectively, on this disease has been likened by Pickering of London to the discovery by Magellan of the straits that bear his name. Cortisone or the adrenocorticotropic hormone exerts a remarkable effect on the group of diseases which Selye of Montreal classifies as the diseases of adaptation. It would be well for psychiatrists to study the steps which have led to Selye's hypothesis, for it includes an evaluation of psychogenic stress. Similar work might solve the problem of the major psychoses, for the fact remains that biochemical and functional pathological factors are unexplored.

In his lecture on hypertension Pickering seemed to think that all psychiatrists insisted on a specific or primary psychogenesis. He discounted the psychogenic factor, because psychiatrists could not tell him the specific nature of the personality disorder responsible. Recently (1950) he has returned to this theme in consideration of Selye's diseases of adaptation and the psychogenic factor in peptic ulcer, rheumatic diseases and ulcerative colitis. He states that the psychogenic hypothesis fails to account for (*a*) the variety of mental states causing the same disease entity, and (*b*) the fact that a single mental state may be associated with a variety of disease entities. Pickering has roused a number of others, including Wells of Liverpool, "who would willingly lend a hand if there is a possibility of interring the psychosomatic hypothesis". Wells attacks the attempts to correlate personality types and diseases such as ulcerative colitis, migraine, hypertension and peptic ulcer. Why must certain schools in medical psychology postulate a pure psychogenesis and ignore the possibility of an equally potent somatic determinant? Such dogmatism jeopardizes the chances that psychiatry may teach a total approach to the patient in medicine and surgery and robs millions of the benefit of a psychotherapeutic approach. The answer seems to be that such "schools" have transformed what was initially a method of treatment or a mode of hypothetical inquiry into a speculative philosophy to bind and nourish the members, while problems of ætiology and therapy remain as before. The ætiologies of Moses, Shakespeare or Francis Thompson can be bought in a book shop, while the mental hospitals become more crowded than ever.

Pickering and other prominent teachers failed to consider the possibility of a non-specific psychogenesis mediated by a specific somatic determinant. Thus the ingredients in external stress or intrapsychic mental conflict may vary considerably in different individuals, but the common result may be a sustained rise in the "diffuse basic anxiety" or "central excitatory state" of the individual, which brings the now well-known physiological mechanisms into play. The final common path of these may be asthma in one person or peptic ulcer in another, depending entirely on local factors—toxic, infective, allergic or genetic. The

psychogenesis may thus play an important but entirely non-specific role. Physicians such as Walshe and Pickering fail to realize the varying significance of the same symptom in psychiatry. Thus the psychiatrist, confronted with dyspepsia for which the physician can find no local or general structural pathological basis, may be dealing with a true anxiety equivalent accompanied by other somatic equivalents such as tachycardia and sweating, and demonstrable radiologically by such findings as pylorospasm. But the same symptom may also be hysterical and provide a compromise solution to unconscious mental conflict; or it may be "conditioned" as in cooks and messmen. Again, dyspepsia may be a true somatic delusion representing the commonest error of the physician or the clinical psychiatrist, for such subjects are depressive psychotics and their lives often end in suicide. More rarely the dyspepsia may be a visceral epileptic equivalent. Sometimes the symptom represents a combination of these reactions, so that the delusional patient's belief is reinforced by the presence of the anxiety equivalent, intensifying his dread and conviction of incurability. The failure to understand the varying significance of a symptom in psychiatry is not necessarily the fault of the physician so much as the hopelessness of the psychiatrist in the matter of definition. Pickering might have considered further that psychogenic stress thus mediated could produce irreversible somatic changes. Had he done so he might have omitted his reference to the failures of psychotherapy. There are many examples in medicine of psychogenic stress producing irreversible somatic changes, from abortions to chronic anxiety states. While it is too late to invoke psychotherapy in the former, its use as the sole method of treatment might be argued for the latter. Yet in those severe long-standing chronic anxiety states in which the appearance of the patient gives rise to the apt term "pseudo-Parkinsonism", the somatic manifestations appear well-nigh irreversible. The response of these subjects to electroconvulsive therapy is sometimes remarkable. Throughout the whole field of psychiatry one of the limitations of a primary psychogenesis is that it could have initiated irreversible structural changes. Hence, Pickering's invocation of the failures of psychotherapy in some psychosomatic disorders to disprove a psychogenesis is not necessarily valid.

The suggestion of a non-specific psychogenesis shaped by a somatic determinant might give rise to the cry of "unnecessary dualism". Yet there is nothing dualistic about the resultant in the parallelogram of forces; and to concede that the end reaction is due to the balanced summation of various factors is the basis of the Adolf Meyer tradition. The Meyer tradition has had a tremendous influence on British and American psychiatry, and one wonders why the text-book edited by Muncie is not more widely read in Australia. The Meyer tradition is a broad total approach to the whole field of psychiatry and is not committed to any particular technique in therapy. To equate it with a particular therapeutic technique is absurd and analogous to a rejection of Osler's approach to general medicine because he did not mention antibiotics. The need for the Meyer tradition has never been greater, for the neophyte in psychiatry finds himself beset by departments and institutes teaching speculative mechanisms before he has learnt the multitude of facts of descriptive clinical psychiatry in the wide and varied field of mental hospital and out-patient clinic. He hears of "guilt and hostility" before he can discern true depression or somatic delusions. Descriptive clinical psychiatry on the widest possible basis is the foundation of psychiatric practice, regardless of the practitioner's leanings in therapy. If the theories of any particular "school" are founded on fact, then it ought to welcome the present trend in the teaching of medicine and surgery to introduce the student to the total life situation of the patient and the descriptive facts of his mental life.

In establishing a psychogenic basis for reactions in psychiatry, emphasis is placed on the sifting of the material produced by the patient. Thus the speech, gestures and drawings of a schizophrenic gathered over a period may provide striking evidence to confirm the hypotheses of a particular school in psychopathology. In fact, a life story of the patient may be constructed with considerable ingenuity to demonstrate a transition from infantile difficulties to the psychotic states. Even if one accepts the validity of the reconstructed pattern, its causal relationship to the psychosis remains unproven. At times the examination of vomitus may reveal the correct ætiology of a gastro-intestinal disorder; but more often its significance is non-specific. Perhaps the point can be made clearer by the quoting of a case which could have had serious medico-legal significance.

The patient was a young man, aged twenty-eight years, an only child of devoted over-protective parents, of German Lutheran stock, and reared in the stilted atmosphere of a past era. He was an intelligent and extremely successful artisan, but had little social life outside his home and no heterosexual interests. His mother was a warm, capacious, managerial woman, who accompanied him wherever he went, even on holidays. The bond between them had been intensified by the development in him of major epileptic fits since the age of twenty years. In recent times the fits had increased in frequency and his anticonvulsant régime was therefore altered. One afternoon shortly after this alteration he left his work and went to his mother asking for his "son". She stated afterwards that he looked confused and that she was certain that he was going to have a fit. However, he declined to lie down and brushed aside the *Sun* newspaper which she thought he had demanded. He then walked out of the house suddenly, while she called his father, who attempted to follow him. The patient walked a considerable distance around the suburb until the father lost him. Then the patient entered several houses until he found one in which there was an infant asleep in a perambulator. He picked up the child and was bringing it home when he was arrested by the police and later taken to a mental hospital. He was quite lucid a few hours later, and in retrospect remembered hearing a voice which he believed to be that of the Holy Ghost commanding him to find the Son. The duration of the psychosis was five or six hours. He had not had any similar episode previously, nor did he have another until the time of his death, which occurred accidentally through misadventure during a seizure while he was on a holiday with his mother. The latter has been too distressed to furnish details.

The formal content of this psychotic episode forms a pattern which permits psychopathological interpretation; but few would deny that the primary cause of his psychosis was psychomotor epilepsy. Yet if the psychotic episode occurred without any suggestion of the epilepsy, would it not be a fact that some medical psychologists would reconstruct a primary psychogenesis? Hence the difficulty with many psychopathological interpretations of ætiologies in psychiatry is that, although they may throw light on the formal content of the psychosis, they may be quite irrelevant to its primary cause. This is exemplified clearly in hypertensive arteriosclerotic psychoses. The formal content of such psychoses is often patterned clearly by the past life of the individual and contains varied topics based on early experiences. In fact, the formal content often permits dynamic psychological interpretation even when the patient has cerebral hæmorrhages, choked disks and uræmia. Hence the problem arises throughout the whole field of psychiatry as to how far the formal content of a reaction can be used to furnish a cause. Is an individual a schizophrenic because the formal content discloses classical material for mental conflict? Or is the role of any mental conflict in schizophrenia simply non-specific? Could the same psychogenesis based on so-called Œdipal, psycho-sexual or aggressive factors produce psychopathy in another case, or merely a successful company director with marital difficulties in still another? There is no doubt that psychopathological mechanisms nearly always contribute an ætiological component of varying importance. But this may be non-specific, mediated physiologically as "stress". Apart from the psychoses, this concept seems to apply to the group of disorders in general medicine, the so-called psychosomatic group.

In recent years the psychosomatic group of disorders has attracted the attention of many physicians and surgeons in various parts of the world because they have felt on clinical grounds that psychogenic factors play a part in disorders hitherto explained in terms of structural abnor-

mality. As a result a chaotic profusion of psychological hypotheses has been poured out into the literature, in which one finds temperamental and characterological traits vying with libidinous investment of various organs for causal linkage with a particular disorder. Thus Wittkower states that in patients suffering from colitis, peptic ulcer and effort syndrome, the common factors are over-conscientiousness and over-scrupulousness. He then differentiates the gastric ulcer patient on a basis of concern with economic security, the colitis patient with over-cleanliness and hoarding, and the effort syndrome patient on the basis of preoccupation with religiosity and patriotism, while Alexander thinks of the patient with mucous colitis "relieving his sense of guilt for oral aggression through the medium of intestinal evacuation".

The net result of this fantastic chaos of thought is that the psychogenic component in medical or surgical illness is discounted and the patient deprived of psychotherapy aimed at resolving mental conflicts and situational or external stresses. Even if psychotherapy deals with situational, conscious or pre-conscious factors, or is confined to non-directive counselling, its value is immense. One of the greatest problems facing psychiatry today is the achieving of a harmonious unity with the general body of medicine and surgery, and there is no greater obstacle to this than speculative interpretations given dogmatically. Meyer's total approach to the patient and his method of distributive analysis are recalled in the concluding words of Walshe's recent oration:

> The medical psychologist must be elective, if he is to preserve his intellectual freedom and integrity, and in the interpretation of human records which are the essential data of his study, his knowledge and critical insight must be dominant instruments.

To alter a phrase of Dr. Samuel Johnson, it may be said that psychiatrists are fair-minded people who rarely speak well of one another. Nothing is less conducive to progress in psychiatry than the hackneyed allegation that another's opinion is due to "an unconscious resistance" to one's own. Psychiatry cannot afford to ignore the views of prominent teachers like Walshe. If a leading neurologist criticizes the psychoanalytic group, its members ought to rebut the criticism with reasoned argument. The opinions psychiatrists express of one another recall an essay of Mr. James Thurber, in which he describes the proceedings at an "intellectual" party, the culmination of which is the "unmasking" of everybody else's "ideology". The invitation which medicine and surgery are at present extending to psychiatry will have to be met with something better than bovinity and mediocrity.

Mention has been made of occasional psychoses resolved by analytical means and elucidated in terms of a primary psychogenesis. One of the greatest difficulties in assessing these cases is the matter of diagnosis. Some of these psychotic reactions are more accurately described as "states", rather than classified as major psychotic disorders, acceptable as such to an experienced alienist. One such alienist had part of his hospital set aside for service psychotics in England during the war. He always referred to this group as the "disturbed neuroses", in contradistinction to his civil patients, who were genuine psychotics. Thus some reactions are more accurately described as paranoidal or schizophrenic states rather than as paranoia or schizophrenia. The same distinction applies to obsessional states in contrast to what may be described as the obsessional psychoses. The psychoses in receiving house material differ considerably in course and prognosis from the chronic psychoses of the mental hospitals, as they do from the drawing-room psychoses discerned on the basis of psychological testing.

In matters of ætiology it is often urged that we know little of constitutional factors, and their invocation is often a resistance to an unacceptable psychogenesis. This is not true. To modern psychology we owe the assessment of intelligence levels and specific aptitudes appearing as inherited factors, as we also owe a knowledge of variations in imagery, perception and energy endowments. The electroencephalogram has clarified and extended the conception of epilepsy as well as that of psychopathic states. There are diurnal fluctuations in energy endowments and variations in sleep as described by Meyer. There are many individual variations in metabolism as in blood-sugar curves and growth factors. The field of physiological psychology is full of promise, for its scope extends to many fields of human endeavour. Kretschmer, Meyer, Jung and others have described the primary types in the spectrum of human reaction types. Despite the intergrades, the schizoid of asthenic hypoplastic physique is demarcated clearly from the cycloid of hyperplastic build. It is insufficiently realized that these types represent extremes of a continuum without being discrete and mutually exclusive. In recent work on physical constitution in women, Linford Rees has demonstrated, as in the male group, a leptomorphic-eurymorphic continuum in a unimodal curve, "the extremes of the curve only, impress as as well marked and antithetical types". There is no doubt that Kretschmer and others over-emphasized these antithetical types, as did Kraepelin in his differentiation of the manic-depressive and schizophrenic group. Yet both these workers were concerned with samples of population in which only moderate diffusion had occurred. Rees's samples were subject to the vigorous diffusion of war service, and his centre was the Mill Hill Emergency Hospital. However, where populations have been static for a considerable period hereditary factors tending to one extreme or the other may predominate. Thus in 1923 the admission rate for melancholia in Inverness was nearly three times that in Glasgow. It is easy for observers to be influenced by the local limitations of their material, and to develop mutually exclusive human reaction types, and mutually exclusive psychotic reactions will inevitably lead to error. It is of interest to note that some genetic studies show an association between manic-depressive psychosis and schizophrenia. The importance of this emphasis on a continuum linking the antithetical types is that the two apparent groups of psychoses may have a common ætiology, the difference in symptomatology being due to the reactions of differing constitutions.

The relationship between bodily structure, mental qualities and type of psychosis has been insufficiently studied, and suffers from the extremes of enthusiasm and prejudice. Statistical studies are often made on limited material. Chronic mental hospital material is particularly neglected, and those ignorant of anthropometric principles are apt to confuse adipose dysplastic schizophrenics with leaner, more heavily boned manic-depressives. Bellak and Holt (1948), pupils of Sheldon, set out the difficulties of such study.

In present-day psychiatry there is a tendency to emphasize the life history of the patient and its interpretation in terms of a psychogenesis and to neglect a detailed constitutional assessment. Yet the results of any type of therapy depend largely on the constitutional assessment, and many lamentable results in treatment are due to neglect of the patient's innate deficiencies and to an assumption that unalterable aspects of his reaction can be modified. Thus cyclothymia fused with a psychoneurotic component in an individual of low intelligence presents a total reaction in which the success of therapy depends on the ability of the psychiatrist to assess the components separately. Psychiatrists could with profit devote much study to the many problems involved in human constitution. Rate of growth and physical maturation is one such problem, and to particularize further, the maturation of the electroencephalographic tracings is of special interest. The tracings of aggressive psychopaths resemble those of young children, as exemplified in the work of Hill and Watterson. Henderson does not despair of some psychopaths until after the age of thirty-five years, for he has noted physical and emotional maturation developing late.

It is in the group of psychoneuroses that the ætiological role of a primary psychogenesis has yielded the most fruitful psychotherapeutic results. However, the disorders in this group do not occur in pure culture as frequently as they do in the form of a psychoneurotic component in medical and surgical conditions, the latter often contributing little to the total incapacitation. As early as

1904 Freud defined the limitations of analytical therapy in the psychoneuroses. He stated that the patient must be intelligent, that he must have a certain amount of education and a certain degree of "ethical development". "Deep-rooted malformations of character, traits of a degenerative condition . . . can scarcely be overcome." Further, the patient must not be too old. If his age is "near or above the fifties . . . the mass of psychical material can no longer be thoroughly inspected, the time required for recovery is too long and the ability to undo psychic processes begins to grow weaker". The patient must be cooperative and consciously desire recovery. Many incidental limitations on psychotherapy are mentioned in some of Glover's writings, such as expense, duration of treatment and the attitude of close relatives. To these perhaps one might add the limitations imposed by compensation or pensions.

Both World Wars have given a tremendous impetus to the study of the psychoneuroses and to a development of psychotherapeutic methods, although there is no consensus of opinion as to the superiority of any particular approach. Glover defines the attitude of the psychoanalytic group in uncompromising style, because he states that "war-time psychiatry has led to a general reaction in favour of superficial (pre) conscious factors in mental disorder", while at the other end of the scale Sargant and Slater emphasize the success of physical methods. Between the dogmatsm of such groups it is possible that the vast psychotherapeutic field open to psychiatry will be lost.

In discussing "The Question of Lay Analysis", Freud makes a number of statements which can be reconciled only with difficulty, if at all. He states that "probably, indeed, no neurosis occurs without some constitutional, congenital predisposition". So perhaps it ought to be stated that the only limitation of a pure psychogenesis is that there is no such thing as a pure psychogenesis. But as one reads on further in the monograph Freud states that "the neuroses of the majority of those who come to us are fortunately psychogenic, and there is no question of pathological elements. Once this has been established by the physician, we may safely leave the analytical treatment to the lay analyst". He states that if the lay analyst has doubt about the origin of symptoms he can appeal to the physician. Freud develops this further and states that if the medical analyst has doubts he has to appeal to a physician too. Presumably the medical analyst has fewer doubts. Then Freud states that "organic and psychical treatments do not go well together in the same hands", so perhaps he does believe in a pure psychogenesis. Of the lay analyst Freud states that "he can find no use for the greater part of what is taught in medical schools. A knowledge of the anatomy of the metatarsal bones, of the properties of carbohydrates, of the courses of the cranial nerves, of all that medicine has discovered as to bacillary infection and means to prevent it, or of serum reactions, or neoplasms . . . all this is of the greatest value in itself, but it will take him nowhere". Freud is hopelessly wrong.

Even in the presence of a relevant psychogenesis, the correct interpretation of symptoms may be a matter of extreme difficulty, necessitating observation over a period of time. Structural abnormality may produce symptoms which are qualitatively indistinguishable from those of a psychoneurosis in pure culture. Freud's error does not rest here. In his paper "Analysis Terminable and Interminable" he states that analysis is "a lengthy business", but even in short-term psychotherapy a further difficulty arises. The symptoms of a psychoneurosis have necessarily an intense emotional significance for the patient. Consequently it is common for the patient to ignore or undervalue other symptoms, of gradual development and sometimes of serious pathological import. The fatigue of mental conflict may merge with that of early tuberculosis, or the early symptoms of a cerebellar cyst may be screened by the long-standing anxiety equivalents of anxiety hysteria. This neglect by the psychoneurotic of early symptoms of somatic disease is a trap for all practitioners because of the readiness with which such symptoms can be fitted into the psychoneurotic picture. Furthermore, the patient unwittingly misleads the practitioner by emphasizing the symptoms of emotional significance. This hazard provides a further limitation upon the concept of a primary psychogenesis.

Conclusion.

Thus in this brief survey the concept of a psychogenesis has been considered in relationship to the general framework of psychiatry, and its limitations, both ætiological and therapeutic, have been considered. The growing recognition by medicine and surgery of the psychogenic component in all illness has led to a scrutiny of what psychiatry has to offer. The confusion of thought as to the role of a psychogenesis has been indicated and a plea for prudent eclecticism urged. In particular the role of a non-specific psychogenesis in terms of "stress" has been introduced.

There are corollaries to this paper concerning the training of psychiatrists and the relationship of psychiatry to the rest of the medical profession, for upon these two aspects the future of the specialty rests. To admit that an ætiology is unknown or that a form of treatment is empirical opens the way to inquiry, and it is in the best traditions of medicine; whereas to call one's speculative philosophy a scientific method and the universal key to diverse human problems means a betrayal of a unique trust.

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NITROUS OXIDE–THIOPENTONE–CURARE ANÆSTHESIA: A PRELIMINARY REPORT ON AN APPARENTLY SATISFACTORY TECHNIQUE.

By Ronald V. Pratt,

Honorary Anæsthetist to Neurosurgical Unit, Honorary Assistant Anæsthetist in Department of Anæsthesia, Royal Perth Hospital, Perth.

Curare plays rather a unique role in anæsthesia. It is not an anæsthetic agent in itself: that is to say, it possesses no central depressant or analgesic properties of any value (Smith *et alii*, 1947); and yet, since its earliest introduction to anæsthetic practice by Griffith and Johnson in 1942, it has been widely and enthusiastically hailed as one of the greatest contributions to modern surgical anæsthesia. Perhaps it is enjoying that first swing of the pendulum and, like many another drug flashing for one triumphant instant across the therapeutic firmament, it may sink presently into the oblivion whence it came; or perhaps its current prestige may be but the beginnings of something greater than we imagine—who knows? Yet for all its enthusiastic reception, its present popularity is far from unanimous and its status is very much in the melting-pot: some will tolerate it in none but a few specially indicated cases, while others must have it on pretty well every conceivable occasion. Perhaps it is as one of the latter that I have clung stubbornly to an earlier conviction that there is in curare a tremendous potential influence, yet to be adequately exploited and developed, for bringing us closer to that ideal anæsthesia for which we have long striven.

Since January, 1947, I have tried quite a number of combinations, both likely and unlikely, of that drug with various anæsthetic agents. The complementary properties of thiopentone and curare—the ideal hypnosis of the one, and the extreme relaxation of the other—presented an obvious anæsthetic affinity which should surely be exploited in some way. Unfortunately, owing to an apparent incompatibility in solution of the two drugs, it scarcely seemed possible at first to combine them into a single agent for intravenous use. It is an undisputed fact that clinical solutions of sodium thiopentone are strongly alkaline, having a pH of around 10·35, and that proprietary solutions of *d*-tubocurarine chloride are on the acid side with a pH of about 5·1. It is also incontrovertible that random mixing of these two solutions will in most cases result in a disconcertingly heavy floccular precipitate of thiobarbituric acid which mere agitation will not redissolve. It appears to be less well known, however, that this precipitate will dissolve in excess of thiopentone solution.

In August, 1947, I began experimenting *in vitro* with solutions of the two drugs, and somewhat empirically determined their solubility limitations imposed by varying concentrations and proportions. Confirmation and much additional information were obtained from a reprint of a relevant paper by Professor Joe W. Baird (1947), and the particular solution was selected (described hereafter as TPC, or thiopentone-curare, solution) which seemed likely to prove the most serviceable one. In a personal communication subsequently received, Professor Baird intimated that the method of preparation of a satisfactory solution finally decided upon by his department was identical with that of TPC solution.

It was about this time that several reports by Professor Ralph T. Knight turned up. He described a specific technique of balanced anæsthesia in which thiopentone, curare and nitrous oxide were employed in certain fixed ratios. One paper in particular seems to summarize with admirable clarity and completeness what has come to be known as "Knight's technique", and well repays careful repeated study (Knight, 1946). The impression made on me was profound, and the technique was used whenever and wherever possible with an ever-increasing enthusiasm and sense of satisfaction. There was only one "catch", however, and that was the necessity to set up for each case an intravenous drip infusion. In attempts to obviate this requirement, several devices were repeatedly tried (such as the use of three syringes in parallel *et cetera*), but none proved satisfactory.

Meanwhile the TPC solution was in a tentative and rather desultory fashion tried in occasional cases during a period of some two and a half years, while other avenues of anæsthetic technique, seemingly more promising, were explored. Such drugs as procaine given intravenously, pethidine given intravenously, and synthetic relaxants, were put to clinical trial and were in turn discarded as unable to fulfil the requirements sought. As the latest product of such efforts to arrive at a satisfactory anæsthetic technique, the scheme outlined below was decided upon and put into daily clinical use early in January, 1950. Originality is not claimed for any fundamentally new technique: it is simply a form of balanced anæsthesia in which practically all factors and components are reduced to constants. This attempted standardization of procedure is the outcome of analyses of previously published techniques, of the selection of what one feels to be the best features of each, and of a resynthesis of these features into a technique subsequently modified by the gradual evolutionary process of clinical usage.

Outline of the Technique.

The method to be described I at present use as a routine measure in most cases, and has been followed meticulously throughout the series presented, except for a very few minor variations recorded in the report.

A solution (TPC solution) is prepared to the following formula: sodium thiopentone one gramme, *d*-tubocurarine chloride 30 milligrammes, sterile distilled water to 40 millilitres.

This is delivered from a 20 or a 30 millilitre syringe, via a length of fine rubber tubing and an indwelling fine needle, into the most convenient vein of the patient (or, in the presence of a continuous intravenous infusion, it may be more convenient to give the fractional injections of TPC solution into the infusion tubing). Induction is effected in this manner, larger doses being given when intubation is to be performed. A pharyngeal airway is then inserted and the facepiece of the gas machine applied (or the endotracheal tube introduced, as the case may be), nitrous oxide and oxygen being delivered at a rate of flow of 500 millilitres per minute of each gas. This flow-rate is not altered until the conclusion of anæsthesia, and the soda-lime absorber is used throughout. To prevent over-distension of the bag, the expiratory valve is opened just sufficiently to maintain the bag adequately filled. Other than normal attention to fundamental anæsthetic principles of airway, respiratory depth (assisted or controlled respiration is at times imperative) and cardiovascular state, there is in broad principle little more to the method than the occasional injection of TPC solution when required by surgical needs or lightening anæsthesia.

Perhaps the principal pitfall in this technique lies in its too obvious simplicity. How fatally easy it is to underestimate our anæsthetic weapons: the disarming facility with which "spinals" or "shots of 'Pentothal'" may be given and all too late regretted! No anæsthetic agent, not even our universally trusted ether, can be given smoothly and satisfactorily unless we have been prepared first to give a good many anæsthetics with it by recommended methods. It has been my personal experience that a strict adherence to the rigid pattern of the nitrous oxide-TPC technique, with scrupulous regard to detail, appears to yield remarkably consistent and satisfactory results—one foots it with easy confidence, as it were, along a well-made road leading securely to one's goal; but when, too early, impromptu variations are practised or short cuts and economies attempted, this solid road too often seems to give out and be replaced by an irregular track winding dismally and without landmarks across some fog-bound swamp.

Discussion of Rationale.

In an attempt to justify this particular anæsthetic technique, I should like to present what appears to me to be the rationale underlying its various component details. Much of this has already been given, and more ably expressed, in Knight's paper (Knight, 1946).

The keynote lies in the phrase "balanced anæsthesia". The ideal anæsthetic should fulfil many criteria. To mention but some, it must provide a pleasant induction and a comfortable aftermath, must exhibit no noxious effect, must give the surgeon all the relaxation and freedom from undesired reflexes in the patient as and when he wishes, must not impede the surgeon's access, must not create concomitant hazards such as that of explosion or fire, and—most importantly—must at all times provide the greatest possible safety for even the worst surgical risks. If possible, too, it should cater for less essential but by no means unimportant considerations, such as simplicity of apparatus, freedom from excessive expense, and ability of a properly equipped anæsthetist to give uniformly smooth anæsthesia with an economy of time, physical strength and anxiety. This ideal anæsthetic yet remains to be discovered. Meanwhile it is possible to select certain existing agents and to combine them in such a way that, while we gain all (or most) of what the surgeon wants in the matter of an unconscious and relaxed patient, we have at the same time reduced the risks to the patient by presenting him with minimal amounts of various drugs whose additive (perhaps synergistic) effects provide adequate anæsthesia with little or no toxicity.

In this technique three drugs—sodium thiopentone, nitrous oxide and *d*-tubocurarine chloride—have been selected to provide between them, after careful premedication, anæsthesia conforming as nearly as can be to the ideal. Each drug is employed for its specific and exclusive contribution to the "balanced anæsthesia". Sodium thiopentone provides the oblivion of narcosis, nitrous oxide contributes analgesia, and curare produces quiescence and relaxation. It is worth while to consider each of these in some detail.

Sodium Thiopentone.

As Knight aptly remarks in more than one of his papers, sodium thiopentone is probably the best hypnotic we have ever had; no other produces sleep as rapidly and pleasantly or allows more pleasant awakening. Unfortunately its excellence stops short at that point: it cannot, and should not, be made to do more than that. Despite the reckless abandon with which countless administrators throughout the world have used it repeatedly for almost every type of surgical operation, it cannot legitimately claim to rank as an anæsthetic agent in its own right. Use it on its own for very short procedures by all means—it is pleasant and convenient and the occasional mishaps will, depending on one's experience and knowledge, probably be few and far between. But do not work it to death or the patient may share its fate. Hart and Weaver (1948), working on rats with several barbiturates (including thiopentone), have stated their conclusions that "the barbiturates are not anæsthetic agents in the best sense of the word, that is, they cannot be relied upon to reduce sensitivity to pain". Again, Himwich (1949) points out that whereas general anæsthetics interfere successfully with the transmission of the nerve impulse by raising the threshold at the synapse, thiopentone and other barbiturates exhibit relatively slight depression of the synapse on the sensory side. All this simply means that, although thiopentone is a wonderful hypnotic agent in safe doses, it cannot provide those conditions of relaxation and of freedom from reflex movement demanded by the surgeon except in much greater doses which, by depressing progressively all parts of the central nervous system down to the medullary centres, impair the organism and endanger life. Thiopentone is also believed to be an ultra-short-acting barbiturate—a rapidly acting and rapidly eliminated drug. While this may approximate to the truth in brief administrations, it does not when large doses have been given; detoxication is delayed and effects tend to be cumulative, approaching those of the long-acting barbiturates. To quote Adams (1947), the problem is no longer one of barbiturate hypnosis or anæsthesia, but one of sublethal barbiturate poisoning with the inevitable depression of respiration, of circulation, and of the nervous mechanisms which control them.

Nitrous Oxide.

Nitrous oxide is probably our weakest anæsthetic agent. Mixed with a safe proportion of oxygen, it may in some cases be scarcely adequate to obliterate consciousness. But for all this it does provide a useful degree of analgesia. The sensory side of the reflex arc being depressed, reflex movements are considerably reduced and this effect is notably enhanced when sodium thiopentone is concurrently given.

While nitrous oxide may be our weakest agent, nevertheless when given properly (that is, with adequate oxygen and ventilation) it is probably our safest, as it is so far as we know completely non-toxic and evanescent in all its actions. It is given in this technique exactly as Knight advocated in his original method, as this appears to me the most satisfactory method I have so far practised. One aims to provide a constant alveolar gas mixture containing 70% nitrous oxide and 30% oxygen. It is held that, if the respiratory exchange is kept at normal levels, 30% of oxygen is entirely adequate for practically all cases (thoracic surgery is beyond my sphere, but I am reliably informed that here, too, with the possible exception of "shunt" procedures, this statement holds good). It is also held that concentrations of oxygen much in excess of this figure are probably unnecessary and may even under certain circumstances provide grounds for ultimate harm.

Now if we attempt to provide this desired alveolar mixture by the orthodox closed circuit method, technical difficulties in maintaining the precise mixture soon arise. Theoretically, having stabilized the mixture in the desired proportions by continuous flow technique, we should be able to carry on thereafter by the sole addition of "basal" oxygen (and provided that we can guess the value of the latter requirement correctly). In actual practice, however, as all anæsthetists are aware, this alveolar mixture is soon upset for various reasons: patients have different and unpredictable metabolic requirements, nitrous oxide may continue to be taken up by incompletely saturated tissues, and the gas may also escape through the skin and the surgical wound; leakage of the carefully stabilized mixture from the anæsthetic system is even more important, and much valuable time and good temper can be lost in attempts to track down the site of the leakage and correct its cause. In administration of anæsthetic agents consisting solely of nitrous oxide and oxygen, it is merely an exercise in applied anæsthesia to restore the *status quo* in the respiratory circuit when such mixture disturbances take place. However, when nitrous oxide and oxygen are given in the presence of sodium thiopentone and curare, I do not see how this can possibly be

done by any clinical yardstick when all the usual signs have been abolished or significantly modified by the drugs given intravenously. It would seem that what we are looking for in this type of balanced anæsthesia is a method of administering nitrous oxide and oxygen so that the alveolar mixture will remain constant irrespective of leakage or diffusion or variable metabolic rate (or even of carelessness on our part), while we retain if possible the economy and other advantages of the closed circuit technique. It would appear that we might be forced back to "continuous flow" anæsthesia, with its wastefulness and difficulty in controlling respiration, to gain at least the constancy we desire; but I believe both from theoretical considerations and from the experience of more than three years' continuous clinical usage that the gas technique described above gives us all these desired features.

From theoretical calculation, one would expect that the result of using a flow-rate of 0·5 litre per minute of each gas, employing the absorber and just enough "blow-off" to keep the bag from over-distending, would, after due flushing out of nitrogen, be an alveolar mixture of 66% nitrous oxide and 33% oxygen (if we remember that 0·25 litre of oxygen more or less is constantly abstracted for basal requirements). In practice, Knight states that repeated analyses of the inhaled mixture disclose an oxygen content varying between 28% and 34%. I have analysed the bag mixture in a series of my own cases and find, when equilibrium is reached, that the oxygen content varies between 28% and 35%, the average for all readings being 33%. As swiftly as the alveolar mixture is upset by minor leakage or other disturbance, so is it replaced by fresh gases which restore the desired mixture before it can be appreciably upset; no adjustments by the anæsthetist are at any time required. Thus one combines the economy of the closed circuit with the invariability of the "continuous flow".

A note of warning may again be sounded on the inadvisability of attempting to apply these principles "with minor modifications". If one reduces each of these gas flows still further, the resultant mixture will be far less dependable and may even result in anoxia; variations in individual metabolic rates will now play an unpredictable and significant part. On the other hand, higher rates of flow, say one or two litres of each gas per minute, will result in a mixture approximating more and more closely to a true 50% mixture of nitrous oxide and oxygen. It may yet be shown that this is a more desirable mixture; but, if it is employed, the anæsthetic results will not necessarily correspond with those recorded in this series; my own preference is still for the setting suggested by Knight.

Curare.

Nitrous oxide-oxygen-thiopentone anæsthesia has been found pleasant and satisfying for many procedures, but relaxation is not usually secured without depressing doses of thiopentone, and troublesome reflexes tend to occur at embarrassingly unexpected moments. Intubation, too, is far from perfectly achieved by its means. Curare seems to be the answer to this problem, and I use it now for practically every type of operation (including such procedures as mastectomy, mastoid drainage, craniotomy, curettage, eye operations, orthopædic and plastic procedures, and many others in which curare is commonly believed unnecessary). In so doing I feel I am considerably reducing the toxicity of the anæsthetic, and especially in very long operations, as well as affording my surgeon ideal operating conditions at all stages; and, paradoxically, it has with lengthening experience appeared to lighten both the responsibility and the labours of the anæsthetist himself. Perhaps the most noteworthy results are the absolute quiescence of anæsthesia and the remarkably prompt recovery of the great majority of patients (in the operating theatre or on the way back to bed) even after some three or four hours of anæsthesia. These results can and do occur with nitrous oxide-oxygen-thiopentone anæsthesia, but unreliably and infrequently.

TPC Solution.

The argument so far attempts to justify the use of these three agents in a system of balanced anæsthesia, and to that point might equally well apply to Knight's technique. In the latter, thiopentone and curare solutions are injected alternately into a continuous infusion in the proportion of 25 milligrammes and 1·5 milligrammes respectively. I believe this original technique, meticulously followed, is from the point of view of reliable and satisfactory anæsthesia an excellent one. Its drawbacks, however, appear to lie principally in (*a*) the need for a continuous infusion, (*b*) the seemingly excessive proportion of curare, and (*c*) the need for many small injections in a sequence which is at times apt to be confusing and therefore far from "fool-proof" in a busy round of operations or in the small hours of the night. For these reasons I believe that a solution which contains thiopentone and curare in the fixed ratio of 50 milligrammes to 1·5 milligrammes, and which can be administered from a single syringe directly into an arm vein, overcomes all these objections.

"I do not believe in rigid combinations of drugs: the anæsthetist should exhibit all the versatility of his calling and employ agents only when and as they are indicated." This statement probably expresses the principal objection with which techniques of this nature are likely to be met. I believe this objection to be valid only under certain circumstances. We remain for the most part earnest students seeking improvements in a field never static but constantly in a state of transition. Such being the case, it surely would seem more profitable to advance slowly but certainly, mastering our newer drugs in set basic combinations before essaying multilateral variations according to an unfounded belief in our powers of improvisation—we should surely learn our scales thoroughly ere embarking upon impromptu symphonies. Consider the reputation for safety and reliability so deservedly borne by ether over the space of a hundred or more years: I very much doubt whether this reputation would persist in such high degree had it hitherto been possible for its countless thousands of users to vary at will its component factors of narcosis, of analgesia and of relaxation. Safety of the patient and skill of the anæsthetist have both, I do believe, been assured by our inability to break down ether to its simplest elements. And so, when some promising successor to this valiant old-timer happens along, let us settle upon what seems on good grounds to be its most efficient form, and adopt this as a basic standardized method from a thorough knowledge of which our next advance may later be made.

Advantages.

The following advantages are claimed for this method of anæsthesia.

Minimal Toxicity.

When oxygenation is assured, neither nitrous oxide nor curare is believed to exert any injurious influence; moreover, all effects produced by them are transitory in nature. Sodium thiopentone appears to be the only potentially cumulative, toxic or dangerous drug present in the technique—and as the average total amount used in this series of operations was less than one gramme, it seems justifiable to assert that the possible toxicity is minimal.

Neither in the series here analysed, nor in that reported by Baird *et alii* (1948), has any significant effect on the cardiac conduction tissues been evident. No disturbances of rhythm or increased myocardial irritability ascribable to the technique have appeared. Circulatory depression has been rare, never so pronounced as to cause anxiety, and due either to the surgical procedure (as in the second stage of the Smithwick operation) or to the anæsthetist's permitting inadequate ventilation; it should not be necessary in these days to remind anæsthetists of the disastrous effects which inadequate respiration, with its all-important build-up of carbon dioxide in the body, can have on the cardio-vascular system.

As for oxygenation, this is at all times adequate—again, if ventilation is not neglected—with a mixture containing from 30% to 35% of oxygen.

TABLE I.

Age. (Years.)	Number of Patients.
Under 20	4
20 to 29	15
30 to 39	31
40 to 49	29
50 to 59	15
60 to 69	11
70 to 79	9
80 to 89	2
Total	116

Universal Applicability.

I believe this technique to be satisfactory for all types of adolescent and adult patients (I have had rather less, though equally promising, experience of the method with children). It was the method of choice equally for the alcoholic and Herculean wharf-labourer (Case II, Table VIII), as for the oldest and most frail subject (Case XXIII, Table IV).

Nor is there, with one or two exceptions to be shortly mentioned, any class of surgery for which it has no place. Despite the apparent irrelevance of curare for procedures not requiring abdominal relaxation, I venture to say that I find the technique as valuable—if not more so—in some of these cases as for intraabdominal operations: craniotomies, for instance, are pleasant and uneventful undertakings (at least from the anæsthetist's cubby-hole) when the "rule-of-thumb" nitrous oxide-TPC method is used.

Non-Explosive Quality.

I have heard eminent anæsthetists addicted to cyclopropane say: "Oh, we don't let our surgeons use the endotherm." Not possessing this degree of eminence, I have always felt it a prime duty to facilitate my surgeon's work to the utmost, and certainly not to impose any avoidable limitation upon his techniques. Having been inflicted with either Knight's technique or the nitrous oxide-TPC method for the past three years, surgeons with whom I habitually work no longer ask if the endotherm may be used. It is used freely, and on unpremeditated occasions, without the least care on the part of anyone for so unnatural an occurrence as an explosion.

Smoothness of Anæsthesia and Ease of Administration.

Thanks to the ever-present curare, at no time is laryngeal spasm the severe and worrying event which it sometimes is with thiopentone alone. In contradistinction, laryngeal spasm is extremely rare, always transient, and so mild as scarcely to arouse comment or action.

Intubation is routinely and rapidly performed whenever indicated; difficulties occur rarely and are due either to an inadequate dose of TPC solution (an event the frequency of which varies inversely with one's experience of the technique) or, more usually, to anatomical difficulties equally formidable in any technique of induction.

The subsequent anæsthesia is remarkable for its smoothness and quiescence, for its minimal disturbance of physiology (to which goal, again, careful observation and extended experience contribute progressively), and for the ease and rapidity with which the optimum light plane of anæsthesia may be controlled while full muscular relaxation is at all times instantly available if required.

Having gradually evolved equipment progressively simplified and specifically adapted to this purpose, I have little hesitation in saying that I find this technique quicker to initiate, easier to supervise, and far less worrying than any other worthwhile method of anæsthesia I have used. Especially is this so when it becomes the elected method in practically all of one's cases: the same standard equipment and solution are used throughout the day, one's manipulations fall into an easy routine conferring mechanical precision and safety, and a series of operations in the same theatre involves no more fuss or preparation at the change-over than substituting a new intravenous tubing assembly and washing the mask and hosing.

TABLE II.

Time. (Minutes.)	Number of Cases.	Average Amount of TPC Solution.	
		Thiopentone. (Milligrammes.)	*d*-Tubocurarine. (Milligrammes.)
0 to 30	5	490	14·60
31 to 60	21	682·1	20·46
61 to 120	63	819·8	24·59
121 to 180	21	903·6	27·10
181 to 240	6	841·7	25·25
241 to 300	1	875	26·25

Rapid and Pleasant Recovery.

Largely owing to the reduced amounts of thiopentone, most patients will respond to questioning within fifteen minutes of termination of anæsthesia. Many will do so before they leave the operating table. A few complain of pain or exhibit mild restlessness, but the majority admit to feeling little or no discomfort (which contrasts with my own experience of cyclopropane). In only one case was "Prostigmin" deemed necessary and given.

I cannot recall any instances of nausea or vomiting in the immediate post-operative period in this series, although both have occurred during the first seventy-two hours in an appreciable number. When vomiting has occurred, in most instances it has consisted of the regurgitation of a cup of tea or glass of water taken some eight to twenty hours after operation. Two practices which I have long followed with apparent benefit in this regard have been the substitution whenever possible both before and after operation of pethidine for the opiates, and the pre-operative advice I give to most patients that they refrain from drinking after operation until they are confident of the absence of nausea. It is tempting to discuss the underlying rationale, but in a report of this nature, hardly permissible.

Complications.

Apart from the well-known effects of careless or inefficient supervision (such as tolerance of a poor airway, of inadequate ventilation, and so on—all of which can in no way be designated as specific to this technique), there appears to be but one source of justifiable annoyance, and that is the occasional difficulty in maintaining patency of the "vein-way". Veins which are difficult to enter, through-and-through puncture of the vein, dislodgement of the needle and needle blockage are the usual causes. An assured "vein-way" is essential to the successful employment of any technique dependent on repeated intravenous injections, and careful observation and thought on this subject well repay any anæsthetist's time; my own experience is that blockage of a needle almost never occurs when I am using my own apparatus, but occurs frequently when institutional equipment is used. With regard to difficulties in venepuncture, this still appears to be largely a matter of practice and attention to minute detail; several times, when arm veins have proved obdurate, puncture of the external jugular vein has provided a quicker and easier approach, and to date no undesirable effects have been observed as a result of this procedure.

Hiccups arise occasionally in the course of upper abdominal procedures when the surgeon stimulates the diaphragm by strong traction. A small amount of cyclopropane may be added to control this complication, but I prefer to add more TPC solution and to control respiration for a brief period.

Table III.

Neurosurgery.

Case.	Patient's Sex.	Patient's Age. (Years.)	Risk Grade.	Operation or Condition.	Time.		Thiopentone. (Milligrammes.)	*d*-Tubocurarine. (Milligrammes.)	Remarks.
					Hours.	Minutes.			
I	M.	35	I	Craniotomy.	4	10	875	36	Pethidine and curare given in addition.
II	M.	25	II	Craniotomy.	2	45	875	26	Pethidine, 25 milligrammes.
III	F.	73	III	Laminectomy.	1	40	650	20	Emergency operation.
IV	F.	25	I	Smithwick, second stage.	2	30	900	27	
V	F.	37	I	Osteomyelitis of skull.	1	15	550	16	
VI	M.	64	II	Transplantation of ulnar nerve.	1	15	1000	30	
VII	M.	53	II	Craniotomy.	4	—	1000	30	Extrasystoles initially.
VIII	M.	21	I	Repair of skull defect.	2	—	900	27	Patient died.
IX	F.	18	I	Craniotomy.	2	30	1000	30	
X	M.	33	I	Craniotomy.	1	45	1800	54	
XI	M.	40	II	Craniotomy.	2	30	1550	46	
XII	F.	36	I	Smithwick, second stage.	2	—	850	25	Pethidine, 50 milligrammes.
XIII	M.	50	I	Laminectomy.	2	30	900	27	Pethidine, 100 milligrammes.
XIV	F.	30	II	Gunshot wound of frontal lobes.	2	30	500	15	Emergency operation.
XV	F.	43	I	Poppen.	2	15	750	22	Extrasystoles.
XVI	M.	42	I	Lumbar disk removal.	1	40	1500	45	Cyclopropane given initially.
XVII	M.	39	I	Craniotomy.	2	20	1250	37	
XVIII	M.	30	I	Craniotomy.	1	15	750	22	
XIX	F.	51	II	Laminectomy.	1	30	625	19	
XX	F.	39	II	Craniotomy.	3	—	850	25	
XXI	F.	39	II	Craniotomy.	—	45	600	18	Case XX, further operation.
XXII	F.	61	II	Craniotomy.	—	45	500	15	
XXIII	M.	14	I	Craniotomy.	3	30	350	10	Difficult intubation.
XXIV	F.	51	II	Smithwick, second stage.	1	45	1050	31	
XXV	M.	47	I	Lumbar disk removal.	1	15	850	25	Gastric dilatation on third day.

Contraindications.

The Incompetent Anæsthetist.

The incompetent anæsthetist is perhaps the principal contraindication. The technique, in the hands of an anæsthetist not prepared to devote careful and continuous supervision to such fundamental details as patency of airway, adequate ventilation with assisted respiration whenever and as required, and physiological sign-posts, is probably as safe as a loaded gun in the hands of a child.

Myasthenia Gravis.

I have yet to encounter *myasthenia gravis* in anæsthetic practice, although it seems feasible that all degrees may occur in the community and that many subclinical cases are met unknowingly in our daily practice. At all events, it seems necessary to exclude curare from any anæsthetic method chosen for a patient known to suffer from *myasthenia gravis*.

Table IV.

Abdominal Surgery.

Case.	Patient's Sex.	Patient's Age. (Years.)	Risk Grade.	Operation or Condition.	Method.	Time.		Thiopentone. (Milligrammes.)	*d*-Tubocurarine. (Milligrammes.)	Anæsthesia.
						Hours.	Minutes.			
I	F.	29	I	Cholecystectomy.	Tube.	1	50	900	27	"Flaxedil", 40 milligrammes.
II	F.	35	I	Abdomino-perineal.	Mask.	4	—	750	22	"Flaxedil", 40 milligrammes; transient shock in third hour.
III	F.	61	I	Cholecystectomy.	Tube.	1	33	650	20	Satisfactory.
IV	F.	71	II	Sigmoid colostomy.	Mask.	—	40	650	20	Satisfactory.
V	F.	24	I	Appendicectomy.	Mask.	1	—	650	20	Satisfactory.
VI	M.	52	II	Volvulus.	Tube.	—	55	750	22	Satisfactory.
VII	F.	70	II	Cholecyst-gastrostomy.	Tube.	1	—	675	20	Satisfactory.
VIII	F.	34	I	Laparotomy, inguinal hernia.	Mask.	1	15	650	20	Satisfactory.
IX	M.	68	III	Laparotomy.	Tube.	—	50	850	25	Pethidine, 50 milligrammes.
X	F.	51	I	Common bile duct explored.	Tube.	1	40	900	27	Satisfactory.
XI	F.	39	I	Cholecystectomy.	Tube.	1	15	650	20	Rise in blood pressure.
XII	F.	39	I	Appendicectomy.	Mask.	—	25	600	18	Satisfactory.
XIII	F.	77	II	Cholecyst-jejunostomy.	Tube.	1	15	600	18	Fall in blood pressure.
XIV	M.	39	I	Gastrectomy.	Tube.	2	20	1000	30	Transient hiccups.
XV	F.	53	I	Cholecystectomy.	Tube.	1	—	650	20	Satisfactory.
XVI	M.	45	II	Cholecystectomy.	Tube.	2	—	1500	45	Difficult intubation.
XVII	F.	68	II	Gastrectomy, cholecystectomy.	Tube.	2	45	850	25	Satisfactory.
XVIII	M.	59	II	Cholecystectomy.	Tube.	1	30	850	25	Satisfactory.
XIX	F.	46	II	Cholecystectomy.	Tube.	1	45	1000	30	Satisfactory.
XX	M.	72	II	Appendiceal abscess.	Mask.	1	45	600	18	Satisfactory.
XXI	M.	47	I	Appendiceal abscess.	Mask.	1	25	1150	34	Cyclopropane at finish.
XXII	F.	58	II	Appendicectomy.	Mask.	1	25	550	16	Cyclopropane substituted.
XXIII	M.	84	III	Bowel anastomosis.	Mask.	1	25	950	28	"Bad risk."
XXIV	F.	76	III	Cholecystectomy.	Tube.	1	45	650	20	Satisfactory.
XXV	M.	45	I	Colectomy, gastroenterostomy.	Tube.	3	15	1100	33	Satisfactory.
XXVI	M.	54	I	Gastrectomy.	Tube.	2	10	950	28	Satisfactory.
XXVII	F.	43	II	Cholecystectomy.	Tube.	2	—	1250	37	Satisfactory.

Table V.

General (Including Orthopædic and Plastic) Surgery.

Case.	Patient's Sex.	Patient's Age. (Years.)	Risk Grade.	Operation or Condition.	Time.		Thiopentone. (Milligrammes.)	d-Tubocurarine. (Milligrammes.)	Anæsthesia.	Post-operative Course.
					Hours.	Minutes.				
I	F.	32	I	Ingrowing toe-nail.	—	18	?	?	Satisfactory.	Satisfactory.
II	F.	32	I	Saphenous ligation.	1	20	650	20	Satisfactory.	Slight vomiting.
III	F.	47	I	Mastectomy.	—	35	375	11	Satisfactory.	Satisfactory.
IV	M.	25	I	Inguinal hernia.	1	—	550	16	Satisfactory.	Satisfactory.
V	F.	?	I	Thyreoidectomy.	?	—	650	20	Unsatisfactory.	?
VI	F.	31	I	Thyreoidectomy.	1	—	700	21	Satisfactory.	Vomiting.
VII	M.	27	I	Inguinal hernia.	—	55	900	27	Satisfactory.	Satisfactory.
VIII	F.	38	I	Radical mastectomy.	2	15	700	21	Undue bleeding.	Satisfactory.
IX	F.	32	I	Mastectomy.	1	10	550	16	Undue bleeding.	Satisfactory.
X	M.	65	II	Inguinal herniæ.	1	—	750	22	Satisfactory.	Satisfactory.
XI	M.	18	I	Inguinal hernia.	—	35	800	24	Satisfactory.	Satisfactory.
XII	F.	52	I	Radical mastectomy.	1	45	500	15	Satisfactory.	Satisfactory.
XIII	M.	21	I	Tibial bone graft.	1	30	750	22	Satisfactory.	Satisfactory.
XIV	F.	18	I	Granuloma in abdominal scar.	1	30	600	18	Satisfactory.	Slight vomiting.
XV	F.	45	I	Radical mastectomy.	2	—	600	18	Satisfactory.	Satisfactory.
XVI	M.	54	I	Osteotomy and nailing of neck of femur.	1	35	750	22	Satisfactory.	Satisfactory.
XVII	F.	76	II	Epithelioma, hand.	—	20	350	10	Satisfactory.	Satisfactory.
XVIII	M.	72	III	Umbilical, inguinal herniæ.	1	35	950	28	Satisfactory.	Bronchitis.
XIX	F.	73	III	Radical mastectomy.	3	15	950	28	Satisfactory.	Slight vomiting. cardiac failure.
XX	M.	? 40	I	Inguinal hernia.	1	20	750	22	Satisfactory.	Satisfactory.
XXI	F.	60	II	Smith-Petersen nail.	3	—	800	24	Vomited once.	Satisfactory.
XXII	M.	23	I	"Crossed leg" graft.	2	—	1000	30	Satisfactory.	Satisfactory.
XXIII	M.	? 40	I	Inguinal hernia.	1	—	650	20	Satisfactory.	Satisfactory.

Asthma.

I do not regard asthma as an absolute contraindication to the TPC technique, but treat asthmatic patients nevertheless with considerable respect.

Thiopentone exerts pronounced parasympathetic effects, which include rapid sensitization of laryngeal and bronchial reflexes so that minimal stimulation of either a remote or a local character may suffice to precipitate dangerous spasm affecting the airway; the administration of atropine or hyoscine in adequate and correctly timed dosage should for this reason never be omitted as a preoperative measure. Curare effects a valuable and considerable reduction in the hazard of laryngeal spasm, but by its action in liberating histamine can initiate varying degrees of bronchospasm. I have seen this a few times (though not in the series under review), and have observed the subsidence of such bronchial spasm after injecting an antihistaminic preparation intravenously; whether the latter was necessary or not I could not say. I employ the nitrous oxide-TPC technique for asthmatic patients, but endeavour to prepare them beforehand with oral doses of phenobarbitone, ephedrine and an antihistaminic drug, in what I hope may be a balanced and logical régime. So far, no evidence of bronchial obstruction during the operation day has appeared in these instances.

Short Surgical Procedures.

The use of the nitrous oxide-TPC technique for short operations lasting perhaps ten minutes or so (as curettage

Table VI.

Gynæcological Surgery.

Case.	Patient's Age. (Years.)	Risk Grade.	Operation or Condition.	Time.		Thiopentone. (Milligrammes.)	d-Tubocurarine. (Milligrammes.)	Anæsthesia.
				Hours.	Minutes.			
I	34	I	Vaginal repair.	1	10	375	11	Satisfactory.
II	61	I	Vaginal repair.	1	30	650	20	Satisfactory.
III	40	II	Vaginal hysterectomy.	1	50	400	12	Satisfactory.
IV	21	I	Bleeding ovarian cyst.	1	—	700	21	Satisfactory.
V	60	II	Vaginal repair.	1	—	625	19	Satisfactory.
VI	41	I	Hysterectomy.	1	45	700	21	Elevated blood pressure.
VII	41	I	Hysterectomy.	1	40	700	21	Satisfactory.
VIII	37	I	Hysterectomy.	1	50	700	21	Satisfactory.
IX	38	I	Hysterectomy.	2	15	1000	30	Sweating.
X	35	I	Rubin's, curettage, cauterization.	—	20	650	20	Satisfactory.
XI	41	I	Vaginal repair.	1	30	1300	39	Satisfactory.
XII	27	I	Vaginal repair, appendicectomy.	2	15	1100	33	Satisfactory.
XIII	47	I	Vaginal repair.	1	50	1000	30	Satisfactory.
XIV	37	I	Pelvic adhesions.	1	—	1000	30	Satisfactory.
XV	48	I	Hysterectomy.	1	40	600	18	Elevated blood pressure.
XVI	52	II	Cystadenoma, bowel anastomosis.	1	20	600	18	Satisfactory.
XVII	23	I	Salpingectomy, vaginal repair.	2	10	1000	30	Satisfactory.
XVIII	45	II	Hysterectomy.	2	—	800	24	Satisfactory.
XIX	36	I	Vaginal repair, hysterectomy.	1	45	650	20	Satisfactory.
XX	39	II	Hysterectomy.	3	30	900	27	Satisfactory.
XXI	49	I	Hysterectomy.	2	15	800	24	Satisfactory.
XXII	30	I	Hysterectomy.	2	30	750	22	Satisfactory.
XXIII	45	I	Oöphorectomy, appendicectomy.	1	10	750	22	Satisfactory.
XXIV	42	I	Vaginal repair.	1	45	900	27	Awkward airway (jaw).
XXV	29	I	Curettage.	—	10	350	10	Satisfactory.
XXVI	51	I	Vaginal repair, hysterectomy.	1	30	650	20	Satisfactory.
XXVII	49	I	Vaginal repair.	2	20	750	22	Satisfactory.
XXVIII	39	I	Vaginal repair.	1	25	750	22	Undue bleeding.
XXIX	35	I	Hysterectomy.	—	55	550	16	Satisfactory.
XXX	35	I	Myomectomy.	1	30	650	20	Satisfactory.
XXXI	26	I	Salpingectomy.	—	50	700	21	Satisfactory.
XXXII	52	I	Vaginal repair.	—	35	600	18	Satisfactory.

TABLE VII.
Genito-Urinary Surgery.

Case.	Patient's Sex.	Patient's Age. (Years.)	Risk Grade.	Operation or Condition.	Time.		Thiopentone. (Milligrammes.)	*d*-Tubocurarine. (Milligrammes.)	Anæsthesia.	Post-operative Course.
					Hours.	Minutes.				
I	M.	67	II	Prostatectomy.	1	15	750	22	Initial fall in blood pressure.	Satisfactory.
II	M.	80	II	Prostatectomy.	1	30	800	24	Rise in blood pressure.	Satisfactory.
III	M.	66	II	Prostatectomy.	1	5	750	22	Satisfactory.	Secondary hæmorrhage.
IV	M.	66	II	Prostatic bed hæmostasis.	1	15	750	22	Rise in blood pressure.	Satisfactory.
V	M.	43	I	Hemi-nephrectomy.	1	30	750	22	Satisfactory.	Satisfactory.
VI	F.	42	I	Nephropexy.	1	25	750	22	Initial fall in blood pressure.	Satisfactory.

or incision of a small abscess) may appear unwarranted. However, it is a procedure which I often adopt in the belief that it is in the patient's best interests, although I am not at the moment prepared to recommend or otherwise defend the practice for general use. Needless to say, one must display meticulous caution in assessing and/or treating any residual curare effect before sending the patient back to bed.

Tonsillectomy.

From experience with the method in a limited number of tonsillectomy anæsthetics for a surgeon who demands the most complete pharyngeal relaxation, I believe that this operation is one of the very few for which the technique is not well suited.

Cæsarean Section.

Obstetric anæsthesia occurs seldom in my practice; the occasional patient for Cæsarean section has usually been given cyclopropane out of deference to orthodox practice in an unfamiliar field. However, one feels that this field may prove to be no contraindication to the technique: a single patient undergoing Cæsarean section for disproportion was recently anæsthetized by the nitrous oxide-TPC method with most encouraging results; 600 milligrammes of thiopentone with 18 milligrammes of *d*-tubocurarine chloride were given over a period of forty-five minutes, the baby cried lustily even before its speedy delivery was completed, anæsthesia could hardly have been more satisfactory, and the patient awoke without discomfort on the table to proceed to a pleasant convalescence.

Poor Veins.

So far as adult patients are concerned, I think the presence of poor veins is a relative contraindication which diminishes almost to vanishing-point as the anæsthetist's skill and experience increase. In the case of small children, I should imagine the technique would on these or similar grounds be contraindicated in many instances.

Analysis of 117 Cases.

I have given over 600 anæsthetics by the nitrous oxide-TPC technique since January 1, 1950. The following analysis is concerned only with the first 117 recorded cases, which was the number to hand at the time when this report was envisaged and commenced.

Premedication.

Premedication tended to follow a fairly regular pattern. For patients of average build and age, one and a half grains of "Nembutal" with one grain of phenobarbitone (or when indicated, two grains of phenobarbitone alone) were usually ordered to be given the night before operation. Then one and a half grains of "Nembutal" were customarily given two hours before operation, followed an hour later by an injection of 100 milligrammes of pethidine with one one-hundred-and-fiftieth of a grain of hyoscine. This basic plan was at times modified according to each patient's supposed needs: "Nembutal" or the analgesic drug might be omitted, morphine might be substituted for pethidine, or atropine for hyoscine. Premedicating drugs actually given on the day of operation have been indicated by relevant initials in the tables.

Age.

Distribution of the 116 patients into age-groups is shown in Table I. The youngest patient was aged fourteen years and the oldest aged eighty-four years.

Average Dosage of TPC Solution Used.

Table II indicates the average total amounts of thiopentone and *d*-tubocurarine chloride actually given for operations grouped according to duration of anæsthetic time. Baird *et alii* (1948) have given a similar table, although my total quantities are less than his in all but the shortest procedures; the reason for this may be the fact that Baird's gas mixture (one litre of each gas per minute) should provide the patient with something like 57% of nitrous oxide, whereas mine gives an approximate figure of 70%. It is feasible to think that my patients may require slightly smaller amounts of TPC solution because they are receiving a slightly more potent gas mixture.

It will be noted that for these operations, lasting at times up to four and a quarter hours, the greatest average amount of TPC solution used contained no more than 0·9 gramme of thiopentone and 27 milligrammes of *d*-tubocurarine chloride.

It will also be noticed that, paradoxically, the table shows smaller average amounts of TPC solution for the

TABLE VIII.
Ear, Nose and Throat and Eye Surgery.

Case.	Patient's Sex.	Patient's Age. (Years.)	Risk Grade.	Operation or Condition.	Time.		Tihopentone. (Milligrammes.)	*d*-Tubocurarine. (Milligrammes.)
					Hours.	Minutes.		
I	F.	25	I	Strabismus.	1	5	700	21
II	M.	34	I	Radical mastoidectomy.	2	—	1850	55
III	M.	47	I	Radical mastoidectomy	2	—	1250	37
IV	F.	36	I	Radical frontal and ethmoid sinus procedure.	2	5	700	21

longer time-groups (three to four hours and four to five hours) than for the two to three hours group. This is almost certainly in part due to the statistically inadequate number of prolonged procedures, and in part possibly to the fact that most of the longer procedures were craniotomies (in which maintenance doses of TPC solution are small and infrequent), whereas the groups of shorter procedures contained more instances of well-built individuals undergoing difficult intraabdominal procedures which required full degrees of relaxation under heavy stimulation. Any worthwhile conclusions regarding average TPC dosage must await a much larger series of cases, in which the time-groups could be further subdivided into the different classes of surgery.

Neurosurgery.

Twenty-five cases have been recorded in the neurosurgical group (Table III). The youngest patient was aged fourteen years and the oldest seventy-three years. With one exception, all were assessed as either grade I or grade II "risks".

Intubation was employed for all patients in this group as a routine measure, the only exception being Case VI (a transplantation of the ulnar nerve). No difficulties were encountered in intubation other than that described in Case XXIII.

The anæsthetic time ranged from forty-five minutes (in two cases) to four hours ten minutes. The smallest amount of TPC solution was used in Case XXIII (350 milligrammes of thiopentone with 10 milligrammes of *d*-tubocurarine chloride), while the largest amount of solution contained 1·8 grammes of thiopentone and 54 milligrammes of *d*-tubocurarine chloride (Case X).

With the exception of Cases V, VI, VIII, X, XVI, XXII and XXV, a continuous blood or saline infusion was set up as a routine pre-operative measure.

Anæsthetic administrations adhered strictly to the pattern laid down earlier in this report, nitrous oxide and oxygen being supplied at the constant rate of 0·5 litre of each gas per minute throughout, with fractional additions of TPC solution intravenously as indicated. No variations on this theme, or addition of other anæsthetic drugs or methods, were made apart from the following: (*a*) an infiltration of the line of incision with up to an ounce and a half of a 1% solution of procaine containing adrenaline (five minims to the ounce), was performed by the surgeon preparatory to all craniotomies and laminectomies; (*b*) from 25 to 100 milligrammes of pethidine were given intravenously in four cases, mostly towards the end of operation; (*c*) an additional dose of curare was given *per se* for intubation in the first case of the group (the series being then scarcely born); (*d*) cyclopropane was given at the commencement in Case XVI, owing to temporary difficulty with the needle in the vein.

It has been observed in most of the craniotomies and laminectomies that, after infiltration with procaine and adrenaline solution, there has commonly been a rise in pulse rate and systolic blood pressure (at times of the order of 50 millimetres of mercury in the case of the latter), which may persist for fifteen or thirty minutes. In one case (Case VII), multiple extrasystoles accompanied this elevation in pulse rate and blood pressure.

In general, all anæsthetic administrations were regarded as satisfactory. It is thought that the blame for any recognizable defects in this group may justly be laid on the anæsthetist rather than on the technique used; such defects comprised undue elevation of blood pressure with vasopressors during second-stage dorso-lumbar sympathectomies, irritable reflexes in Case XI, momentary cyanosis in occasional instances, and an acutely embarrassing moment of coughing during a deep craniotomy, shortly after a member of the operating theatre staff had unwittingly emptied out my entire stock of TPC solution.

The majority of these neurosurgical patients were awake or responding before they left the operating theatre, or when examined shortly afterwards in their rooms. Apart from compression cases, post-operative vomiting of any consequence was conspicuously absent, and the subsequent post-operative course in all cases in this group (except Case VIII, in which extradural bleeding was followed by death) was recorded on adequate follow-up studies as completely satisfactory from the anæsthetist's viewpoint.

Abdominal Surgery.

There are 27 cases in the abdominal surgery group (Table IV). The oldest patient was aged eighty-four years, while the youngest was aged twenty-four years; six patients were aged over seventy years. Three patients were regarded as "poor risks" (Grade III).

It will be noted that 18 patients were subjected to upper abdominal surgery; this surgical field is taken as a firm indication for intubation, which was therefore carried out in each of the 18 cases. All other patients were anæsthetized under a face-mask.

The anæsthetic times ranged from twenty-five minutes to four hours. Total amounts of TPC solution used varied from 550 milligrammes of thiopentone with 16 milligrammes of *d*-tubocurarine chloride to 1500 milligrammes with 45 milligrammes respectively.

Conduct of anæsthesia conformed at all points with the exact technique described earlier, except for minor additions in five cases as shown in Table IV. When this table was first compiled, there was an additional column for "post-operative remarks"; this has been deleted, as every case had been recorded as "satisfactory" excepting Case XXI (in which ileus and a fæcal fistula developed subsequently).

General, Orthopædic and Plastic Surgery.

The general, orthopædic and plastic surgery group (Table V) consists of 23 patients whose ages ranged from eighteen to seventy-six years, and includes two patients classified as "poor risks" (Grade III). In only one case (Case XIX) was intubation employed.

The anæsthetic times varied between eighteen minutes and three and a quarter hours. The smallest amount of TPC solution required contained 350 milligrammes of thiopentone, while the maximum amount used contained 1000 milligrammes (one gramme).

With the single exception of Case V, in which a deputizing anæsthetist substituted cyclopropane, there were no variations whatever from the technique reported on.

Gynæcological Surgery.

The gynæcological surgery group (Table VI) comprised 32 patients aged between twenty-one and sixty-one years. There were no "poor risks", and on the whole anæsthesia tended to be smooth and satisfactory at all points. Intubation was not resorted to excepting in Case XVI, in which upper abdominal malignant disease had been suspected pre-operatively.

Except for the administration of 100 milligrammes of pethidine in Case IX, the described technique of anæsthesia was followed rigidly in all cases. The maximum amount of thiopentone used in a single case was 1·3 grammes. For all uncomplicated vaginal repairs, the average amount of thiopentone was 772 milligrammes, while that for hysterectomy was 718 milligrammes.

Genito-urinary Surgery.

The genito-urinary surgery group is a small one of six patients, comprising four men and one woman (Cases III and IV represent operations on the same man). Ages varied between forty-two and eighty years, the average age being sixty.

In none of these cases was intubation used, and apart from some unimportant fluctuations in blood pressure, the group behaved completely satisfactorily from the anæsthetist's viewpoint. The two patients who had an initial and transient fall in blood pressure (to 110 and 100 millimetres systolic, respectively) behaved in this regard as do many patients whose anæsthesia is induced by thiopentone.

It is interesting to observe that in all but one case, exactly 750 milligrammes of thiopentone and 22·5 milligrammes of *d*-tubocurarine chloride were required (in Case II 800 milligrammes of thiopentone were required). Using TPC solution and nitrous oxide, we find a comparatively narrow range of variation amongst patients so far as thiopentone requirements are concerned, which is not seen when thiopentone alone (or even combined with nitrous oxide) is employed.

Ear, Nose and Throat and Eye Surgery.

The four patients in the ear, nose, throat and eye group were young and "good risks". Intubation was used for all. Anæsthesia and the subsequent post-operative course were in each case satisfactory in all respects.

Case II: The patient, a man, aged thirty-four years, weighing 15 stone, was a muscular wharf-labourer generously addicted to alcohol. It is therefore not surprising that, even after heavy premedication, he should provide the record for the largest total amount of TPC solution required in the whole series. This was 74 millilitres, which contained 1·85 grammes of thiopentone and 55·5 milligrammes of *d*-tubocurarine chloride.

Summary.

One hundred and seventeen cases in which anæsthesia to a pre-determined plan was administered with nitrous oxide and a solution containing thiopentone and *d*-tubocurarine chloride have been presented. The patients' ages ranged from fourteen to eighty-four years, and the risks were assessed as grade I, II or III.

Duration of anæsthesia varied from ten minutes to four hours and ten minutes. In forty-four cases (37·6%) intubation was required, difficulty being experienced in two cases. The course of anæsthesia was in general regarded as satisfactory apart from occasional errors in routine anæsthetic technique. Hiccups occurred in one (or two) cases, sweating occurred in one, three patients manifested increased bleeding in the operative field sufficient to draw the surgeon's comment, and one patient presented signs of transient shock believed to be surgical in origin.

Recovery of consciousness was in nearly every case prompt and usually with little or no distress. "Prostigmine" was given in one case. Post-operative nausea and vomiting were uncommon and almost never of a serious nature. Other post-operative complications were rare and difficult to ascribe to the specific technique; gastric dilatation occurred in one patient on the third day, one hypertensive cardiac patient showed diminished reserve for several days after an unduly long operation, and two patients developed minor respiratory infections.

There was one death in the series, due to cerebral compression and not believed attributable in any respect to the anæsthetic.

Acknowledgement.

Acknowledgement is made to Professor Ralph T. Knight and Associate-Professor Joe W. Baird, of the University of Minnesota, to whose stimulating work the technique discussed in this report owes the greatest part of its existence.

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Reviews.

VIRUS AND RICKETTSIAL DISEASES.

An immense amount of investigation of viruses and rickettsiæ has been carried out in the last fifteen years, and the purpose of "Virus and Rickettsial Diseases" is to make the fruits of this available to the general medical reader.[1] It may be said at once that this purpose is admirably fulfilled. Clear and succinct accounts are given of the rickettsioses, thirty-eight virus diseases of man, and bacteriophage. The four authors are well qualified to write on these subjects, for each has contributed notably to their investigation.

With every disease emphasis is given to the characters of the virus, the diagnostic aids which the laboratory can offer, the epidemiology, and methods of control when useful methods are available. The description is rounded off by a brief but adequate outline of the clinical features and treatment.

The opening chapters deal generally with the nature of viruses, their physical and chemical properties, the natural history of virus disease, the reaction of cells to invasion, immunity and chemotherapy. A balanced account of the main features of the rickettsioses is given in 57 pages, and there follows a chapter on the psittacosis-lymphogranuloma group of viruses.

With smallpox the virus laboratory can be of the greatest assistance to the health officer, for there are four reliable laboratory tests—microscopic demonstration of virus in stained smears of the vesicle, inoculation of chorio-allantois, serological tests for antigen in lesions and for antibody in serum. Many will be surprised to learn that the vaccinia universally used for smallpox prevention is not cowpox. Jenner began vaccination with cowpox, but at some time since in some obscure way a modified strain of smallpox which we know as "vaccinia" became substituted. Varicella and zoster are considered to be different manifestations of the same virus. The term "zoster" is used without the prefix "herpes" to avoid confusion with *herpes simplex*—a quite different disease.

The account of influenza includes the present status of attempts at control by vaccination, and the same chapter deals with the common cold, febrile catarrh and primary atypical pneumonia. Measles, rubella, mumps, glandular fever, virus gastro-enteritis are dealt with in turn. A useful table summarizes the properties of, and the differences between, the viruses of infective hepatitis and homologous serum hepatitis.

The various forms of arthropod-borne virus encephalitis are described. The known causes of benign lymphocytic meningitis are listed (lymphocytic choriomeningitis, mumps, herpes, glandular fever, leptospirosis *et cetera*), and then it is stated that the ætiology of the majority of cases of this syndrome in England is at present unknown, but a virus is probably concerned. Poliomyelitis is fully discussed, and after the evidence for the rival theories of spread from throat secretions or intestinal excretions is set out, the conclusion is arrived at that "the most likely source and mode of entry of the virus will depend upon the prevalent ecological conditions".

The book closes with a valuable table showing for the rickettsioses and fifteen virus diseases precisely what diagnostic help the laboratory can afford in each, the material required for the tests and the time taken before the result is available.

The book does not entirely escape criticism: "Mediterranean fever", used (page 63) as a synonym of *fièvre boutonneuse*, is not a desirable term as it invites confusion with brucellosis; DDT is not to be recommended as a

[1] "Virus and Rickettsial Diseases", by S. P. Bedson, M.D., F.R.C.P., F.R.S., A. W. Downie, D.Sc., M.D., F. O. MacCallum, B.Sc., M.D., and C. H. Stuart-Harris, M.D., F.R.C.P.; 1950. London: Edward Arnold and Company. 8½" × 5½", pp. 396, with some illustrations. Price: 24s.

miticide (page 100); "proper" clothing, although desirable for general military hygiene, affords practically no protection against scrub typhus unless impregnated with a miticide, for the mite is small enough to penetrate it (page 100); the dog is not the natural host of *Dermacentor andersoni* (page 103), though it is of *Dermacentor variabilis*; the statement (page 107) that no satisfactory tick repellent has yet been found overlooks or discounts reports of the use of dimethyl phthalate and other substances; misspellings of Ilheus (page 277) and *papatasii* (page 341) are noticed. These points are mentioned for correction in a future edition.

In conclusion the book well fulfils its endeavour to provide a coherent readable account, compact rather than exhaustive, of the virus and rickettsial diseases. References are adequate. The book is to be heartily recommended, especially to physicians, health officers and clinical pathologists, and is likely to have a wide circulation.

CHILD HEALTH.

THE "Handbook of Child Health", by Austin Furniss, contains much valuable information on school medical work in England that is not readily obtainable elsewhere.[1] Although intended primarily for students preparing for the diploma in child health, it is of considerable value to all who are interested in the health of school children. Extensive references are made to dental and mental problems, but this is essentially a book for medical officers. Being thoroughly up to date and based on the personal experience of the author, who is chief assistant school medical officer, West Ham, it can be recommended to anyone who is desirous of knowing what is being done in England. In spite of the ravages and upsets of the last war the provisions for dealing with the minor conditions found among children are far more extensive than anything found in Australia. Undoubtedly the greater population of the country and the size of the larger cities make this possible, but even for the less common conditions boarding schools have been established where children from several districts with diverse complaints can be grouped for study and treatment.

Special emphasis is laid on the need for suitable activities for bed-ridden children whose formal education may occupy only a few hours each day. This handbook would be of value to those who are considering an extension of school medical services, since it includes all the conditions likely to occur in England and shows what has been done. The school dental service in England presents staffing problems, just as it does here, and apparently the only solution is the training of an adequate professional staff for this work.

Unfortunately, the proof-reading has not been up to the standard one expects in a text-book, so that some spelling errors occur. In spite of these the handbook is well worth reading.

X-RAY DIAGNOSIS IN PÆDIATRICS.

THE second edition of "Pediatric X-Ray Diagnosis: A Textbook for Students and Practitioners in Pediatrics, Surgery and Radiology", by John Caffey, has been received.[2] The first edition appeared in 1945 and the present well-arranged volume is enlarged and contains 842 pages with 1039 illustrations and index. To each chapter a bibliography is appended.

The work is intended to give good descriptions and illustrations of the normal and morbid tissues with a proper evaluation of radiographic appearances in clinical diagnosis. The systems are considered regionally, the head and neck being dealt with first and the bones and joints last.

The appearances of the skull at various stages of development are described carefully with notes on variations. A useful table lists the various ossification centres with times of fusion of the bones. The author points out that congenital dysplasias occur mainly in the medial plane. Meningocele and lacunar anomalies as well as traumatic lesions are well illustrated and described. Many rare and unusual conditions are included.

In children intracranial pressure causes widening of the sutures. Intracranial pneumography is stated to be a simple procedure—the technique being fully dealt with—and the findings are most helpful.

Lesions of the thoracic wall and intrathoracic structures are illustrated and described most fully and the author stresses the importance of correct tube centring in obtaining films.

The defects of the diaphragm which lead to herniation of the abdominal viscera are dealt with in detail.

In regard to the respiratory tract, a section on fœtal respiration is included. The anatomy of the lungs in children is well described. Pulmonary tuberculosis is due to inhalation of bacilli and any part of the lung may be affected. The Ghon focus is formed in early life and frequently clears, leaving the well-known calcified areas. Thymic lesions are particularly well illustrated. A good deal of modern work is included in the section on cardiac lesions.

The gastro-intestinal and biliary tracts are adequately covered. The author reports a case of gall-stones in a child of two and a half years.

In the investigation of the urinary tract excretion pyelography has proved most satisfactory. It is contra-indicated when renal function is poor.

To the average practitioner the section on the bones and joints is probably the most interesting part of the book. The descriptions of the normal appearances of the soft tissues and bones is most thorough. Lipomata and abnormal calcifications are dealt with in detail and the illustrations are excellent.

The section on development of bones and the description of the ossifying centres are most complete, and various defects in developments which might be mistaken for pathological lesions receive mention. The author arranges the diseases of the bone in an orderly manner without the usual efforts at rigid classification.

The author's experience in all sections of bone pathology is most extensive, and no other text-book can approach this for the personal knowledge acquired over the many years of his association with various pædiatric hospitals. A feature of the work is its illustrations, and as a book of reference it is in a class by itself.

THE HEIGHTS AND WEIGHTS OF BOYS AND GIRLS.

A. SUTCLIFFE, M.A., B.Sc., Chief Education Officer, the County Borough of Lincoln, and J. W. Canham, M.A., science master at the City School, Lincoln, are the authors of a book entitled "The Heights and Weights of Boys and Girls",[1] which is based on a mass of statistical information obtained in a careful survey of the physical measurements of a great many school children from large towns and small towns and rural areas which it is claimed forms a scientifically representative cross-section of English school children.

The charts and tables are clear and commendably simple, but the textual matter varies considerably from over-simplification, which is at times tedious, to the statement and description of concepts in a manner which provides the reader with some very indigestible material. The mathematical treatment used by the authors is quite valid from the point of view of pure mathematics, but the method of interpretation and the actual use of the figures obtained are not considered to be valid so that any conclusions deduced from the authors' indices are liable to be erroneous.

To substantiate this criticism it is necessary to explain briefly how the indices are derived and then to illustrate how they are used and to mention some of the fallacies. In order to get a true picture of the results of the survey, the authors had to measure variations from the mean figure for a given height, weight and age by compiling "indices" varying in the same manner as the normal distribution of the population as sampled; thus the indices are not the more usual linear functions of the variations of which most of us have had experience.

Reference to the foot-note on page 29 will confirm that the authors' conception of an index to measure, for example, relative height is claimed to be analogous to the determina-

[1] "Handbook of Child Health", by Austin Furniss, L.R.C.P., L.R.C.S. (Edin.), L.D.S., D.P.H. (Univ. Manchester); 1950. London: Sylviro Publications, Limited. 7¼" × 4¾", pp. 284. Price: 25s.

[2] "Pediatric X-Ray Diagnosis: A Textbook for Students and Practitioners of Pediatrics, Surgery and Radiology", by John Caffey, A.B., M.D.; Second Edition; 1950. Chicago: The Year Book Publishers, Incorporated. 10" × 7", pp. 892, with many illustrations. Price: $22.50.

[1] "The Heights and Weights of Boys and Girls", by A. Sutcliffe, M.A., B.Sc., and J. W. Canham, M.A.; 1950. London: John Murray. Melbourne: Geoffrey Camberlege, Oxford University Press. 10¼" × 7", pp. 90. Price: 18s. 3d.

tion of intelligence quotients and to be capable of somewhat similar usage. The method, briefly, is to take a sample which is typical of the population at a certain age and arrange the individuals comprising the "sample" in order; if there are one hundred individuals in the sample, those of average height are rated at height index 50 and the others are given indices above and below according to position on the normal distribution curve. No allowance is made for divergent developmental rates, types of body build or variations in nutritional states. In pursuance of parallelism between physical measurements and intelligence tests, the authors have propounded the idea of a height-age which is defined as the age corresponding to the chronological age of the average child of a certain height; the height-age of a boy is the age of the average boy of the same height; and the weight-age of a boy is the age of the average boy of the same weight. These can be very misleading conceptions in that they relate particular individuals to an average without taking all the pertinent factors into account; such relationships do not tell the full story about any individual and great care needs to be taken in interpreting them. The authors go on to propound a height-quotient by expressing the height-age as a percentage of the chronological age; the height-quotient may show that the individual is taller or shorter than the average at his age, but does not help in the appraisement of his nutritional state or growth. They have given some consideration to the measurement of the natural variations in weight for height due to age and type of body build and have postulated and calculated girth-height quotients using an "index of chubbiness"; chubbiness varies with age and diminishes with increasing years; the index is based for girls and for boys on the rating of the average boy at thirteen years as quotient 100. The conclusions seem to depend on the initial premise that girth is a measure of body volume; that can be only a crude fraction of volume; chest depth and shoulder width have claims which are at least as good as that of a girth measurement which will vary from hour to hour and be measured differently by successive examiners.

In conclusion, we consider that the sampling methods to obtain the data are reasonable, but the arrangement and use of the data severely limit the application of the figures for the general and medical purposes of the physical examination of school children. The same field has been covered much more adequately and satisfyingly by others. The book is stimulating and of considerable interest to any who are seriously concerned about the need for improvement in the technique and utility of routine physical examinations of children.

PSYCHIATRY FOR NURSES.

"A MANUAL OF PSYCHIATRY" by Dr. K. R. Stallworthy is a welcome addition to the many recent similar text-books which aim to provide at the same time instruction for the psychiatric nurse-in-training and an introduction to psychiatry for the medical student.[1] Such a bilateral approach only too often makes such a book too academic and unintelligible for the former, and too laden with mental hospital routines for the latter. Whilst not completely escaping either Scylla or Charybdis, the present volume succeeds in providing a more balanced approach to the subject than most of its fellows.

The book follows strictly orthodox lines. A short chapter dealing with the mind in health precedes a discussion on abnormal psychology—which unfortunately does not escape the over-simplification so common in such books. The causes of psychiatric disturbances are discussed much too briefly for the needs of the student. The following chapter dealing with mental hygiene is admirable, and is followed by a very practical discussion of the general management of psychiatric patients. Short chapters are devoted to the psychoses, neuroses, epilepsy and mental deficiency. The vexed question of psychopathic personality is discussed, but the author carefully avoids any definition of such a state and appears to regard it as a potpourri of conditions that otherwise escape classification. The chapter on special methods of treatment discusses the practical problems of electroconvulsive therapy, electronarcosis, insulin therapy, prolonged narcosis, malarial therapy and leucotomy.

The book on the whole is noteworthy for the clarity of its exposition; it eschews any attempt at discussing theoretical aspects and should prove an adequate text-book for the psychiatric nurse for whom it is intended.

[1] "A Manual of Psychiatry", by K. R. Stallworthy, M.B., Ch.B.; 1950. New Zealand: N. M. Peryer, Limited. 5" × 7¾", pp. 308. Price: 25s.

LIGHT THERAPY.

"LIGHT THERAPY", by Richard Kovács, is a clear exposition of our present-day knowledge on the subject.[1] This lecture (as Dr. Kovács himself designates it in his preface) covers, the physical and physiological effects of the various wavelengths employed in the administration of infra-red, visible and ultra-violet radiation, with details of dosage and technique. Indications and contraindications for the use of ultra-violet radiation, the apparatus and the clinical conditions have been discussed. The whole is well illustrated with photographs and diagrams. This brochure should be of great use to all medical practitioners, as well as to physiotherapists.

[1] "Light Therapy", by Richard Kovács, M.D.; 1950. Illinois: Charles C. Thomas. Oxford: Blackwell Scientific Publications, Limited. 8¼" × 5¼", pp. 120, with illustrations. Price: 16s. 6d.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The Art of Scientific Investigation", by W. I. B. Beveridge: 1950. London, Toronto and Melbourne: William Heinemann (Medical Books), Limited. 8¾" × 5½", pp. 190, with a few illustrations. Price: 10s. 6d.

An introduction to research.

"The Science of Heredity", by J. S. D. Bacon, M.A., Ph.D.; 1951. London: Watts and Company. 6½" × 4¼", pp. 200, with many illustrations. Price: 3s. 6d.

Intended for the general reader.

"Your Children's Feet", by Charles A. Pratt; 1951. London: Watts and Company. 7¼" × 4¾", pp. 62, with fourteen illustrations. Price: 7s. 6d.

Deals with the feet of children from thirteen to fifteen years of age.

"The 1950 Year Book of Drug Therapy (October, 1949-September, 1950)", edited by Harry Beckman, M.D.; 1951. Chicago: The Year Book Publishers, Incorporated. 7" × 5", pp. 578, with many illustrations. Price $5.00.

One of "The Practical Medicine" series of year books.

"Cybernetics: Circular, Causal, and Feedback Mechanisms in Biological and Social Systems: Transactions of the Sixth Conference, March 24-25, 1949, New York, N.Y.", edited by Heinz Von Foerster; 1950. New York: Josiah Macy, Junior Foundation. 9" × 6", pp. 212. Price $3.50.

The subjects discussed included psychological moment in perception, neurotic potential and human adaptation, the quantum theory of memory, possible mechanisms of recall and recognition, and the sensory prosthesis.

"Metabolic Interrelations: Transactions of the Second Conference, January 9-10, 1950, New York, N.Y.", edited by E. C. Reufenstein, Junior; 1950. New York: Josiah Macy, Junior Foundation. 9" × 6", pp. 280, with many illustrations. Price: $3.95.

A conference of twenty-five persons discussed eleven subjects dealing with different aspects of bone metabolism.

"Renal Function: Transactions of the First Conference, October 20-21, 1949. New York, N.Y.", edited by S. E. Bradley; 1950. New York: Josiah Macy, Junior Foundation. 9" × 6", pp. 176, with many illustrations, some of which are coloured. Price: $2.50.

The subjects discussed included "The Morphological Aspects of Renal Tubular Secretion and Reabsorption", "Role of Glutaminase in Tubular Processes", "Biological Aspects of Renal Tubular Transport", "The Tubular Secretion of Potassium and Acid", and "Water Reabsorption by the Renal Tubules".

The Medical Journal of Australia

SATURDAY, APRIL 7, 1951.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

COMPULSORY CHEST RADIOGRAPHY IN WESTERN AUSTRALIA.

In September, 1949, attention was drawn in these columns to the newly introduced *Tuberculosis Act*, 1949, of Tasmania. This Act provides for notification by a medical practitioner to the Director of the Tuberculosis Division of the Department of Public Health of any person who "is or may be" suffering from tuberculosis, or who is revealed by post-mortem examination to have been suffering from tuberculosis at the time of his death. The notification is required to state whether the disease is in active form or whether the patient is in an infectious condition. On the receipt of notification the Director is empowered to have certain things done. He may require the medical examination of all persons who have been in close association with the patient. If the Director is satisfied that the patient is in an infectious condition, he may apply to a Board, and the Board may in certain specified circumstances order the patient to be removed to an institution "where he can be properly attended and treated". Further, the Minister may, by notice published in the *Gazette*, require persons over fourteen years of age to undergo radiological examination of their lungs. This will not apply to any person who holds a certificate from his medical attendant or from an officer of the department to the effect that within a period of twelve months preceding the gazettal of the notice he has undergone a radiological examination of his lungs. It was pointed out in discussion of this measure that the Tasmanian Branch of the British Medical Association had been approached by the Legislative Council of the Tasmanian Parliament in regard to the provisions of the measure. The Branch Council recognized that in the control of tuberculosis some degree of compulsion was necessary, but had expressed the opinion that generally speaking it was opposed to any compulsion of the individual in medical matters.

The Government of Western Australia has, during recent weeks, introduced an Act to amend the *Health Act*, 1911-1948. The Act now becomes the *Health Act*, 1911-1950. The chief clause in the amended Act to which attention should be drawn is as follows:

6. The principal Act is amended by adding the following section:

293A. (1) The Commissioner may, by notice published in the *Gazette*, require all persons over the age of fourteen years of any class or classes specified in the notice to undergo X-ray examinations for tuberculosis at such times and places as are specified in the notice and all persons to whom the notice applies shall, subject to subsection (3) of this section undergo the examination accordingly.

(2) The Commissioner shall cause a copy of the notice promulgated pursuant to the provisions of the last preceding subsection to be published once at least in not less than three newspapers circulating in the State and may in addition cause the contents of the notice to be made known to the public in such other manner as he considers desirable.

(3) The provisions of subsection (1) of this section shall not apply to a person of a class in respect of which class a notice is promulgated pursuant to those provisions, if that person is the holder of a certificate signed by a medical practitioner by whom he has been professionally attended or by an approved medical officer certifying that the person has within a period of twelve months immediately preceding the date of the publication of a notice in the *Gazette*, undergone an X-ray examination of his lungs; but the person shall if required in writing to do so by the Commissioner produce the certificate and the report of the radiologist by whom the examination was carried out to the Commissioner or to a person specified by the Commissioner.

Before the introduction of the Bill into the Western Australian Parliament, Dr. J. Gordon Hislop, who is a member of the Legislative Council, wrote to the Council of the Western Australian Branch of the British Medical Association asking for its views. The Executive Committee of the Council considered the question purely from the medical aspect. It expressed the opinion that the X-ray examination of chests for the discovery of tuberculosis was desirable. It considered that such examination should be compulsory for all migrants, and that there should be identification photographs to ensure that the person submitted to X-ray examination was actually the migrant concerned. While the Executive Committee agreed that X-ray examination of the community was a good thing in respect of the control of tuberculosis, it stated that the question of compulsion was not a medical one, but a political issue. These views were sent in a letter to Dr. Hislop, and the same letter was sent to the Minister for Health. When the Bill came before the House, Dr. Hislop and others opposed strongly the compulsory provisions of the Bill. Their protests were of no avail, and the Act has been passed.

At this stage it will be useful to draw attention to a recently published article by E. G. W. Hoffstaedt, entitled "The Pattern of Mass Radiography".[1] Hoffstaedt deals with the cost and the limitations of mass radiography. He reports on the work in the north of England of four units, three of which were completely mobile and independent; they took between them 97,250 miniature films during a year. When all the expenses and running costs of the unit were taken into account, the average cost per miniature film worked out at 4s. 6d. Cases in which further examination was necessary as a result of suspicious findings in the miniature film numbered 6247, or 6·4% of the total number. Large films taken in these cases worked out at a cost of £3 8s. 10d. each. With a figure of 4s. 6d. as a basis, Hoffstaedt uses the incidence rate of pulmonary tuberculosis discovered by mass radiography to estimate the cost of each discovery. When the incidence rate is 2%, the highest which in Hoffstaedt's opinion may be found, the cost per case was £11 5s. The cost increased until, with an incidence rate of 0·35%, the cost per case was £64 10s. These are the figures for an average survey; but Hoffstaedt shows

[1] *The Lancet*, December 9, 1950.

that the cost may be much higher. In one survey of 53,790 persons undertaken in a certain area over a period of two and a half months, only two active infections previously unknown were discovered. The cost of the survey was £1264, and thus the cost in each of the newly discovered cases was £632. He holds that mass radiography of the total population on a voluntary basis cannot even be attempted. The units and personnel at his disposal could not, he declares, deal with more than a small fraction of the adult population. One unit can manage from 25,000 to 30,000 miniature films a year. The five units at his disposal are able to deal in one year with 7% of the adult population of the region. It is intended in future that routine surveys shall be made in hazardous industries, especially dusty occupations like mining, sand-blasting and steel dressing, in industries and factories known to have an unusually high incidence of tuberculosis, such as tobacco and clothing factories, especially those employing young women, and in any factory or workshop in which an unusual proportion of employees are known to have developed tuberculosis. It is also proposed to have quarterly sessions at each main centre of population, at which will be examined groups of persons which he enumerates. *The Lancet* asks editorially whether an attempt is not being made to use radiography in the wrong way. It approves of the selected groups enumerated by Hoffstaedt.

Attention may also be drawn to a report issued by the Medical Research Council of the Privy Council of Great Britain in 1945 (see The Medical Journal of Australia, June 23, 1945, page 641). The title of this report was "Mass Miniature Radiography of Civilians for the Detection of Pulmonary Tuberculosis". The authors of the report were Kathleen C. Clark, P. D'Arcy Hart, Peter Kerley and Brian C. Thompson. In this report the results of a survey of 23,000 persons are given. An example quoted in our reference to the report deals with 5200 available employees. Of these 90% attended for examination. Of the 90%, 5·5% were recalled in order to have large films taken, and of this fraction 2·5% were further medically investigated. Tuberculosis of significant type and degree was disclosed in 1·5% of the total, and treatment was found to be necessary in 0·4%. In addition a number of non-tuberculous lesions were discovered.

Readers of this journal are well aware that surveys of the population by miniature radiography are not of themselves sufficient to reveal tuberculous infection. In view of the amendment to the Western Australian Act, however, which deals with radiography only, the statement must be made again. The work on this subject done by such persons as Reginald Webster, of Melbourne, and published in this journal will be recalled. The first conclusion to be stated is that emphasized by the Executive Committee of the Council of the Western Australian Branch—namely, that X-ray examination of members of the community is a desirable step in the control of tuberculosis. When this is carried out, provision must be made for further investigation of persons whose miniature films show suspicious markings. This was done in the work reported by Hoffstaedt, and in that recorded in the Medical Research Council's report in 1945. If this is not done, it is likely that some persons will be branded as suffering from active lesions and be subjected to unnecessary psychological stress. It will be remembered that tuberculosis was discussed at the plenary session of the Perth Congress in August, 1948. H. W. Wunderly, in dealing with case finding, said that case-finding surveys should be conducted, even if there was a shortage of beds. He held that if infective persons could be located, and by education and treatment made non-infective to those with whom they came in contact, the medical profession would have gone a long way towards stopping the spread of tuberculosis. This is no doubt true, but in order to make these persons non-infective hospital beds will often be necessary. It must be presumed that the Western Australian Government is seized with the necessity of providing adequate beds for tuberculous patients who need them. Many persons will agree with Hoffstaedt that mass radiography of the total population cannot even be attempted on a voluntary basis. To do so under compulsion would be still more difficult. The point is that when the Act is a "drag-net" Act no one can complain that invidious distinctions are made against persons of certain occupations and in certain strata of society. It has been proved over and over again that more persons suffering from tuberculosis will be found in some groups than in others. We may presume that, although the Commissioner in Western Australia will have the power to call up all persons over the age of fourteen years for radiological examination, he will not necessarily do this. His activities will most likely be restricted to groups of persons who, for one reason or another, are likely to be the subject of tuberculous infections. He will probably adopt this procedure firstly because of his own understanding of the magnitude of the undertaking, but also no doubt because of the difficulty which he will experience in obtaining the necessary staff and equipment, and possibly also because of the enormous expense which is likely to be entailed. The only aspect of the subject which remains to be mentioned is that of compulsion. On this divergence of opinion may be expected. On the one hand those who oppose compulsion will think that explanation and persuasion should be sufficient and that to compel a person to submit to X-ray or other examination is an encroachment on his liberty of action. Against this the compulsionists put up the sound argument that pulmonary tuberculosis is spread by sufferers who are apparently healthy, in other words that tuberculosis is an infectious disease, and that if compulsion is used against other common infectious diseases there is no reason why an exception should be made with tuberculosis. In favour of this view is the undeniable fact that a condition cannot be treated unless it is known to exist. The important consideration that must be emphasized, whether compulsion is used or not, is that examination must be complete—that miniature radiography is not sufficient, but must be followed by complete bacteriological examination when any doubt exists. Everything will depend on the wisdom with which the new Act in Western Australia is administered; its working will be followed with the greatest interest.

Current Comment.

AN INDEX OF TUMOUR CHEMOTHERAPY.

Investigations into the treatment of cancer have been many and diverse; and especially in the field of chemotherapy, they have very often resulted in little or no

success. It is of great importance that investigators should have available to them the results obtained by others if important clues are not to be lost and if blind alleys are not to be repeatedly followed. For the individual worker this has been impossible because of the vastness of the associated literature, and the announcement will therefore be welcomed that the National Cancer Institute in the United States of America has prepared and issued an index of tumour chemotherapy.[1] This is a monograph of 329 pages, compiled by a biochemist of the Institute, Dr. Helen M. Dyer; it is described, quite justly one would say, as the most comprehensive survey made to date of the literature on the results of treatment of tumours by chemical methods. It is intended to provide cancer research workers with a single source from which the available information on this subject can be obtained, and contains data from the chief reports on tumour chemotherapy published in the American, English, French, Japanese, German, Italian, Portuguese, Scandinavian and Spanish scientific literature. It is not exhaustive—many practical factors including the imposition of a reasonable time limit prevented that—but it is as complete a survey as possible in the circumstances. Nor is it critical, pertinent data having been tabulated without discrimination as to their relative value; but again it was not practicable to make it otherwise. The report has four main sections: a historical résumé and explanatory introduction; a table of 5031 chemotherapeutic tests classified according to the chemical used; an alphabetical index of the chemicals, provided with keys so that they may be instantly located in the table of chemotherapeutic tests; a bibliography of the 2213 references from which the data were taken. The table of chemotherapeutic tests contains, for each chemical, data on the type of tumour, the species and number of animals used, the dosage, the number of treatments, the route of administration, the effect claimed, the year the test was reported, and the reference. Attention is drawn in the foreword to the conclusions of the Council on Pharmacy and Chemistry of the American Medical Association published in 1949. The Council states that the established treatment for cancer at present consists of the judicious use of surgery and irradiation. It concedes that some promising discoveries offer hope that specific therapy may some day become of value, but until further research has been completed, the use of drugs must, for all practical purposes, still be regarded as experimental. These conclusions, the foreword points out, should be kept in mind in evaluating the data presented in the index.

The number of copies of this report are limited, and its distribution is being confined chiefly to persons engaged in cancer research work. However, in order that everyone to whom the report may be of service may have an opportunity to use it, copies have been sent to all medical schools and other large scientific libraries and to several large city libraries in the United States and Canada, and to many libraries in countries outside North America. Requests for copies will be granted to the extent possible within the limitations imposed by the size of the edition and the relative needs of groups and individuals. Requests should be addressed to the Cancer Reports Section, National Cancer Institute, Bethesda 14, Maryland, United States of America.

[1] "An Index of Tumour Chemotherapy: A Tabulated Compilation of Data from the Literature on Clinical and Experimental Investigations", by Helen M. Dyer; 1949. Washington: National Institute of Health, United States Public Health Service. 9" × 11¼", pp. 336.

ACUTE BARBITURATE POISONING.

ACUTE barbiturate poisoning provides a problem in treatment that may confront any medical practitioner. For some time now investigators have appreciated the effectiveness of the analeptics picrotoxin and "Metrazol", but have withheld final judgement on their right use and on their potential harmfulness. In an authoritative report on analeptics by J. E. Eckenhoff *et alii*, to which reference was made in these columns on August 27, 1949, the conclusion was offered that picrotoxin and "Metrazol" were the most valuable analeptics currently obtainable; picrotoxin was the most potent and also the most dangerous to use and should be used only in deep depression and then by someone well versed in its actions. Before the true part played by these drugs in the treatment of depression could be fully evaluated, the report stated, more complete and detailed clinical summaries must be made and compiled. T. Koppanyi and J. F. Fazekas[1] have set themselves to compile the required summaries and to analyse the cases of barbiturate poisoning published in recent years.

Koppanyi and Fazekas have dealt with reports of 88 cases of human barbiturate poisoning, culled from papers published in the past fifteen years; they have deliberately omitted papers from which no instructive or practical information could be extracted. The cases have been placed in six groups according to the barbiturate involved (barbital, phenobarbital, "Amytal", pentobarbital, "Seconal" and mixed barbiturates) and each group is analysed separately. The major conclusion drawn is that picrotoxin exerted a life-saving action. No conclusive evidence was found to indicate that amphetamine, "Coramine", disodium succinate and caffeine exerted such an action. It appeared that many patients with acute barbiturate poisoning who were treated with analeptics did not require specific treatment and would have recovered with adequate symptomatic treatment. Koppanyi and Fazekas are not satisfied that the eligibility of a patient for analeptic therapy can be determined by inspection of the apparent degree of coma present; in their opinion the best indication is the response of the patient to an orientation dose, a single intravenous injection of five millilitres of 10% solution of "Metrazol". Some patients, although to all appearances in an extremely deep (Grade IV) stage of barbiturate coma, awaken immediately after such a dose. Patients on the other extreme may show signs of only barely noticeable respiratory stimulation. The orientation dose should never be given unless there is presumptive evidence that the patient is poisoned with barbiturate or some other aliphatic narcotic. If there is uncertainty, the "Metrazol" should be given slowly and the administration stopped if twitches appear about the body. If the patient responds favourably to the orientation dose, "Metrazol" should be the agent of choice; its administration should be continued in a dosage of five millilitres of 10% solution every five or ten minutes until complete awakening results. If the patient does not react favourably to the first dose or to repeated doses of "Metrazol", picrotoxin therapy should be instituted. An initial dose of 25 milligrammes is given by vein, with a further amount of 15 milligrammes in a similar manner every fifteen minutes until there is evidence of either complete awakening or at least a return of reflexes. Then the dose of picrotoxin should be reduced, but medication should not be discontinued until the patient is able to perform purposeful, voluntary movements. Koppanyi and Fazekas consider that in most cases "Metrazol" is the drug of choice for the ultrashort-acting, short-acting and intermediate-acting barbiturates. For poisoning with massive doses of short-acting and intermediate-acting or with long-acting barbiturates, picrotoxin is the drug of choice. Several objections sometimes raised to analeptics are dismissed by these investigators in relation to barbiturate poisoning, but their warning should be heeded that central stimulants are contraindicated in comas produced by central nervous system depression other than that caused by barbiturates or other aliphatic hypnotics. It is plain that these analeptics, especially picrotoxin, are not in every way ideal agents for the treatment of barbiturate poisoning, and measures to supersede them should be sought. Nevertheless, they are of value and are the best we have; Koppanyi and Fazekas have indicated how they may be more effectively used.

[1] *The American Journal of the Medical Sciences*, November, 1950.

Abstracts from Medical Literature.

PATHOLOGY.

Cancer of the Cervix Uteri.

HERBERT L. LOMBARD AND EVELYN A. POTTER (*Cancer*, November, 1950) state that the strong correlations that exist between cancer of the uterine cervix and marriage before the age of twenty years, divorce or separation of partners at any time, unrepaired lacerations, the birth of the last child to women before the age of twenty-five years, and syphilis indicate that these variables are of ætiological significance. There is some question whether or not late onset of puberty and douching with coal-tar derivatives are of importance. Poverty and racial incidence are mentioned as of probable significance, though this is not substantiated by this study. The reasons for the various correlations are not clear, although there is a suspicion that infections, chronic irritation, and hormonal imbalance are of considerable importance.

Primary Diffuse Tumours of the Meninges.

BOYD K. BLACK AND JAMES W. KERNOHAN (*Cancer*, September, 1950) present four cases of diffuse meningeal meningiomatosis. They state that evidence supports the fact that there are two types of cells in the meninges. In two of the authors' cases, tumours appear to have arisen from meningothelial cells, one from fibroblasts, and one of mixed type from both meningothelial cells and fibroblasts. This suggests that both elements of the meninges may undergo diffuse neoplasia. The cases were in children or young adults, as have been most cases reported in the literature.

Local Gigantism (a Manifestation of Neurofibromatosis): Its Relation to General Gigantism and to Acromegaly Illustrating the Influence of Intrinsic Factors in Disease when Development of the Body is Abnormal.

KEITH INGLIS (*The American Journal of Pathology*, November, 1950) states that local softening of bone is due to the influence of the neural intrinsic factor of neurofibromatosis or of a basic intrinsic factor. Osteochondromata of the phalanges associated with macrodactyly are due to the influence of neural intrinsic factor of neurofibromatosis (or basic intrinsic factor) acting locally, and are not due to faulty control by the nervous system in the ordinarily accepted sense, that is, by neurons and axons. When elongation of a limb is associated with extensive hæmangioma in the affected part, the elongation of the limb is not caused by excessive vascularity and increased blood supply, but the hæmangioma and the elongated overgrowth have a common underlying factor, namely, neural intrinsic factor (or basic intrinsic factor). When local gigantism occurs in a patient with general gigantism or with acromegaly, the local enlargement is due to neural intrinsic factor of neurofibromatosis, the general enlargement being due to the hormone of the anterior lobe of the pituitary gland. The lesion of the pituitary gland responsible for general gigantism or for acromegaly is predisposed to by the influence of intrinsic factor, probably at the basic intrinsic factor level. Concomitant lesions of acromegaly, such as local gigantism, *cutis verticis gyrata*, syringomyelia, lipomata, and neurofibromatosis, are not due to the hormone of the anterior lobe of the pituitary gland, but are of the same order as the pituitary lesion; intrinsic factor (neural or basic) underlies them all, including the pituitary lesion.

Elastic Tissue, Calcium and Arteriosclerosis.

H. T. BLUMENTAL, A. I. LANSING AND S. H. GRAY (*The American Journal of Pathology*, November, 1950) describe how the hepatic, renal and iliac arteries of approximately 140 human subjects have been studied by means of routine hæmatoxylin and eosin and elastic tissue preparations, and by microincineration; a comparison has been made of the age changes in these vessels and those in the aorta and coronary artery as previously reported. The present investigations reaffirm the authors' previous conclusion that the basic age change in the major arteries of man is primarily calcification of the media. These studies show that the calcification is intimately associated with alterations in the physical character and pattern of distribution of the elastic tissue, which are described; the intensity and rate of calcium deposition are directly proportional to the intensity and rate of the elastic tissue changes. It has been demonstrated further that the location of these processes within the wall is different in different vessels, as is also the rate of calcification. The iliac artery calcifies most rapidly, followed by the renal and coronary arteries, which show an almost identical rate of calcium deposition. The aorta and hepatic artery calcify relatively slowly. An attempt has been made to correlate certain disease processes with the intensity of the age changes. In general, the number of cases of a specific disease in a given age group is too small to warrant any conclusion, but there appears to be an intensification of the aging processes in the hepatic artery of individuals with diseases such as cirrhosis and carcinoma of the liver which may obstruct the hepatic blood flow. These observations yield additional information on the life history of elastic tissue. The term elastoid is suggested for the fragments and granules of elastic material which increase in the aging process of arteries, pending further knowledge of their physical and chemical characteristics.

Vascular Degeneration in Hypothyreoidism.

WILLIAM B. KOUNTZ (*A.M.A. Archives of Pathology*, December, 1950) describes how an anatomical study of four persons whose thyreoid glands had been completely removed for therapeutic reasons and a study of 13 other persons, seven of whom had low rates of oxygen consumption, showed medial degenerative changes in the aorta and larger blood vessels. In contrast, five of the 13 persons who had normal average basal metabolic rates for a five-year period before death and one who had a moderate increase in the rate of oxygen consumption did not show this change. Attention was paid to the thyreoid gland at autopsy. In the four patients whose thyreoids had been removed, no evidence of thyreoid tissue was found. In those who had a low basal metabolic rate, frank degenerative changes were present in the thyreoid, and these substantiated the clinical impression of low function of this gland. In each instance in which there was diminished thyreoid function, medial arterial changes were noted in the blood vessels. In the younger persons whose thyreoid glands had been removed and who had lived for a five-month period after the operation, areas of cystic degeneration were noted. Another person, the one who lived for two and a half years and took thyreoid for a part of the period, showed advanced degenerative cystic necrosis. The one who lived for five years after thyreoidectomy and received no thyreoid had advanced arteriosclerosis and medial degeneration. The author states that this work reveals that hypothyreoidism and its associated metabolic deficiency in many may lead to advanced degeneration of the blood vessels when present over an extended period.

Non-Specific Shock in Experimental Poliomyelitis.

G. M. FINDLAY AND E. M. HOWARD (*The Journal of Pathology and Bacteriology*, July, 1950) state that in mice infected intracerebrally with the Lansing strain of poliomyelitis virus an intravenous injection of either "T.A.B." vaccine or diphtheria toxoid, or of pertussis vaccine *plus* diphtheria toxoid, causes a more rapid onset of paralysis and earlier death. The differences in the interval between infection and death in control and in vaccine-treated mice were significant in five out of six experiments. These findings are discussed in relation to the precipitation of poliomyelitis in man by non-specific stimuli, paralysis being most severe in the limb which has been stimulated.

Giant-Celled Tendon-Sheath Tumours and Related Conditions.

H. SPENCER AND I. W. WHIMSTER (*The Journal of Pathology and Bacteriology*, July, 1950) state that the two conditions of giant-celled synovial tumour and cutaneous histiocytoma, although differing widely clinically, nevertheless possess many features of similarity in their pathology. The giant-celled tumours of both joints and tendon sheaths can be found to originate from a chronic inflammatory condition of a synovial membrane. A similar sequence is thought to occur in cutaneous histiocytoma. Both conditions pass through a series of changes commencing as a chronic inflammatory lesion and progressing through a proliferative cellular phase to end by becoming a mass of fibrous tissue. The histological features of the phases through which both lesions evolve have many similarities. In the early stages the basic chronic inflammatory lesion is characterized by the presence of numerous thin-walled capillaries. Later, as the cellular proliferative phase is reached, both conditions show giant cells, lipoid-laden cells, macrophages containing iron pigment, and proliferation of the stromal cells. In this phase evidence of the inflammatory nature of the conditions is provided by the endarteritis and periarteritis of the smaller vessels

and by the presence of scattered collections of lymphocytes in the lesion. Finally, both types of lesion become hyalinized fibrous masses, and it may then be difficult to realize that these were related to the earlier stages. The minor differences in structural detail which have been observed between the lesions of the two groups are probably attributable to the differences in the stroma of the two tissues in which the lesions arise. It is of interest also to note the essential similarity in structure between the giant-celled epulis and the two conditions described above, for the inflammatory nature of the former lesion has been accepted by many.

The Ovarian Brenner Tumour.

JAMES W. REAGAN (*American Journal of Obstetrics and Gynecology*, December, 1950) states that the Brenner tumour is an uncommon benign ovarian neoplasm which is usually unilateral in location. The solid or cystic tumours are variable in size, usually firm, pale yellowish-grey, and well circumscribed. A study of their histopathology reveals polyhedral cells arranged in masses and columns frequently surrounded by degenerate and hyalinized connective tissue. When cysts occur, they are lined by flattened, cuboidal or columnar epithelium containing glycogen and secretory granules staining with mucicarmine. In the author's study of 23 cases, serial sections of a small subcortical tumour revealed definite continuity between superficial inclusions and the tumour. A second tumour studied did not reveal conclusive evidence of such an origin.

MORPHOLOGY.

Human Cerebral Tumour Containing Five Fœtuses.

D. L. KIMMEL *et alii* (*The Anatomical Record*, February, 1950) describe a tumour of the brain of a nineteen-day hydrocephalic infant which contained representative parts of five fœtuses. Two of the fœtuses are distinct individuals; the other three are represented only by extremities. The structure of the tumour, the body form and skeletal development of the five fœtuses, and the microscopic structure of one of the fœtuses are described.

Age Changes in Direction of Mental Foramen.

R. WARWICK (*Journal of Anatomy*, April, 1950) records the course and direction of the mental foramen as observed in the human mandible in different stages of its growth from fœtus to adult. In the adult, the canal which connects the foramen with the inferior dental canal curves not only laterally, but also markedly upwards and backwards, so that the foramen points upwards and backwards and presents a grooving or smoothing-out of its margin in the postero-superior quadrant. At birth, however, the grooving of the foramen margin is inclined upwards and forwards. The change from newborn to adult configuration is acquired in the first few years of life. Almost all foramina after the age of five years point at least upwards, but usually backwards as well, whereas in the newborn they pointed always upwards and forwards. From observations on the comparative anatomy of the foramen, the author finds that when single and therefore comparable with recent human condition, the foramen is directed upwards and forwards, a fact which may be related to chinlessness or alveolar prognathism. However, the growth changes which produced a chin do not seem to provide adequate explanation of the change in direction of the foramen, and it is concluded that the functional factor of jaw movements is also involved.

Structure of the Liver.

H. ELIAS (*The American Journal of Anatomy*, November, 1949) submits, as a sequel to his previous description of the laminar structure of the liver, the present study concerning the manner in which the laminæ and the labyrinth compose the lobules and how they are correlated with the circulatory and biliary systems. The lengthy account is based on a variety of methods, including injection methods, wax reconstruction from serial sections and the indigo-carmine (excretory) method. Human post-mortem material was utilized only to verify, as far as possible, *a posteriori* the findings in experimental animals. The article is illustrated with stereograms, photomicrographs and diagrams.

Intramural Blood Supply of Human Upper Jejunum.

F. S. DORAN (*Journal of Anatomy*, July, 1950) has studied radiographically the first loop of the jejunum with its mesentery, removed from 12 adult bodies at autopsy. The distribution of the *vasa recta* and their main branches is demonstrated and also that of the smaller arteries and arterioles. The arterioles tend to be arranged in parallel rows lying inside the two mucosal leaves of each circular fold. The relative avascularity of the antimesenteric border is confirmed, but it is shown to be true of only a certain number of bodies. The circumferential anastomoses of the terminals of the *vasa recta* from opposite sides of the bowel are demonstrated. Longitudinal anastomosis is poor and depends upon small vessels. The efficiency of this system is demonstrated by radiographs prepared after ligation of the *vasa recta*. It is found to be variable, and at best to be able to bridge a gap of two and a half to three inches.

Spinal Trigeminal Nucleus.

J. OLSZEWSKI (*Journal of Comparative Neurology*, June, 1950) finds that, in contrast to the usual description, the nucleus of the spinal trigeminal tract is not uniform in structure, and cannot be regarded as an uninterrupted continuation of the posterior cornu of the spinal cord extending up to the main sensory nucleus in the pons. He states that the so-called spinal trigeminal nucleus can be subdivided on the basis of its cytoarchitectonic structure into three parts—*nucleus caudalis*, *nucleus interpolaris* and *nucleus oralis*. Only the *nucleus caudalis* has the same fundamental cell arrangement as the head of the posterior horn of the spinal cord; the *nucleus interpolaris* and *nucleus oralis* have quite different structures. According to these anatomical findings, we may consider that only the *nucleus caudalis* is concerned with the conduction of pain and temperature from the face. The connexions and functions of the two other nuclei are for the time being unknown. These results explain why the operation of tractotomy is effective, even when the section is made as far caudal as at the level of the obex. It is suggested that the term spinal trigeminal nucleus should be avoided and, instead, one should speak of the spinal trigeminal complex, which is composed of three nuclei: *caudalis*, *interpolaris* and *oralis*. The spinal trigeminal complex has the same general features and the same subdivisions in both man and monkey.

Supradiaphragmatic Vagus Nerves from Ulcer Patients.

E. A. GASTON AND C. G. TEDESCHI (*Archives of Neurology and Psychiatry*, October, 1950) have subjected to histological study the supradiaphragmatic portions of the vagus nerves removed at operation on 21 patients with duodenal ulcer and also some removed at autopsy. The study showed in a number of instances a gamut of lesions, including inflammatory cell reaction, myelin degeneration, neurolysis, neuronal atrophy, and perineural and intraneural fibrosis and scarring. These changes were found approximately twice as frequently in patients with duodenal ulcer as in patients without ulcer. It is assumed that lesions of the peripheral portions of the vagus nerves might, in some circumstances, play a part in the development of disorders of structure or function occurring in segments of the alimentary tract under vagal control.

Structural Changes in Nerve Cells following Excitation.

C. N. LIU *et alii* (*Journal of Comparative Neurology*, April, 1950) have performed experiments on guinea-pigs and cats to determine whether or not structural changes in nerve cells could be produced by electrically induced activity in neurons. Comparison of sections of control and experimental guinea-pigs failed to reveal any change of Nissl pattern in the cells of spinal ganglia and spinal cords after prolonged electrical stimulation when adequate precautions against post-mortem and fixation artefacts were observed.

Cell Ratios in the Visual System.

K. L. CHOW *et alii* (*Journal of Comparative Neurology*, April, 1950) have made three independent estimates of the number of neurons in the lateral geniculate body and in the striate cortex of two specimens of *Macaca mulatta*. The average of the three determinations of the total number of cells in the lateral geniculate body was about one million and in the striate cortex about 145 millions. These figures and other data allow estimation of the cell ratio of geniculate to receptive cortical lamina, of separate macular and peripheral geniculostriate ratios, and of the proportion of cells in striate cortex representing macular vision. Postulated mechanisms of visual acuity and of preservation of visual engrammata are discussed in relation to these estimates. Existing comparative data on numerical relationships within the visual system are summarized to indicate a possible quantitative approach to the problem of visual acuity and to emphasize the need for more precise information.

Special Article.

MURRAY VALLEY ENCEPHALITIS.[1]

A severe human encephalitis of virus origin spread diffusely along the Murray Valley during the early months of 1951. This has been provisionally referred to as Murray Valley encephalitis (MVE). The virus of Murray Valley encephalitis resembles that causing Japanese encephalitis B, and in nature has infected horses and possibly dogs in addition to man. In view of the interest that has been evoked in the public Press and elsewhere by the recognition of this disease at the same time as and in the same areas as have been involved in the spread of the rabbit virus disease myxomatosis, it has been thought desirable to present a brief account of the investigations that have so far been completed. These show conclusively that there is no relationship whatsoever between the two diseases. A full account of clinical, field and laboratory investigations will be published at a later date.

Earlier epidemics of encephalitis have been described in Australia by Cleland *et alii*. Outbreaks occurred in 1917, 1918, 1922 and 1925. The 1917-1918 outbreaks of what was known as Australian X-disease were investigated by Cleland, Campbell and Bradley (1919), who showed that infection could be experimentally transmitted to monkeys and sheep. The virus was not maintained, and there has been no opportunity of establishing its relation to the encephalitis viruses now known.

Early in February, 1951, information was received by the Institute indirectly from Dr. H. A. Lanyon, Medical Superintendent at Mildura Base Hospital, that several patients with a severe form of encephalitis had been admitted to his hospital during the previous two weeks. A few days later a similar report was received from Dr. Michael Benson, Medical Superintendent at Mooroopna Base Hospital, regarding three cases arising in the Shepparton district. Further inquiry revealed a minor prevalence of the same type of encephalitis in Victoria along the Murray Valley and its tributaries from Beechworth to Mildura. By March 20, 1951, 40 cases had been studied. All had been severe and their clinical picture had varied around a mean which might be described as follows. Typically the condition commenced with irritability and headache, followed next day by nausea and vomiting, with fever to perhaps 105° F. The patients when admitted to hospital generally exhibited a moderate degree of neck stiffness, and some were irrational to the point of violence. Cerebro-spinal fluid taken at this time contained approximately 100 cells per cubic millimetre (variation from 20 to 750 cells was encountered). The cells comprised polymorphonuclear leucocytes and lymphocytes with sometimes up to 10% of monocytes. The protein content of the cerebro-spinal fluid was sometimes slightly raised; the chloride content was reduced in some cases to below 700 milligrammes per 100 millilitres; the sugar content was generally either normal or just a little below normal. During the patients' first few days in hospital tremors and localized spasticity were noted in many cases and the patients became semicomatose. At the end of a week or ten days deepening coma and complete flaccidity of the muscles preceded death in many cases. Other patients slowly improved and eventually completely recovered.

Of the 40 patients so far studied, 23 have been aged less than twelve years; the ages of the remaining patients have been scattered fairly evenly between twelve and seventy-five years. Thirty of the patients were males.

The geographical distribution of Murray Valley encephalitis approximately follows the distribution of the population in those areas of the Murray Valley in which there is abundant water. The Shepparton and Mildura irrigation areas have produced the majority of cases. Although several cases of encephalitis have been noted in the Melbourne area, there is yet no serological evidence that they were due to the Murray Valley encephalitis virus.

On February 8 a male patient, aged nineteen years, died at the Mooroopna Base Hospital. Central nervous tissue was brought to Melbourne on dry ice (solid carbon dioxide) and emulsions were inoculated into several species of laboratory animal and developing chick embryos. Five days later the virus, when in its second egg passage, produced focal lesions on the chorio-allantois of the embryos. On transfer to mice it produced a rapidly fatal encephalitis. The virus is being maintained both by chorio-allantoic passage in chick embryos and by intracerebral passage in mice. It produces no symptoms or lesions in rabbits and has no serological relation to the virus of myxomatosis.

A serological test became practicable following on the finding that a saline extract of infected chorio-allantoic membrane provided a good complement-fixing antigen. A complement-fixation test of this type has been found to be quite suitable for a survey of sera. For example, of 21 clinical cases the patients in 19 have exhibited a high titre of complement-fixing antibody to Murray Valley encephalitis virus during convalescence. In many cases early death has prevented serological confirmation of the clinical diagnosis.

Careful inquiry has been made for the occurrence of minor illness which might represent mild infections by the Murray Valley encephalitis virus. In a study of 16 such cases no serological evidence has yet been obtained to suggest that such cases are recognizable.

Serum was examined from 109 people, each of whom had been living in association with a patient suffering from Murray Valley encephalitis. Eleven of these contacts had a level of antibody in their serum which suggested previous infection with the virus. The clinical nature of the cases, their distribution and the nature of the virus all suggested strongly that Murray Valley encephalitis was due to one of the insect-borne encephalitis viruses, of which the best known are Japanese encephalitis B, of Japan and adjacent areas, and the equine encephalitides and St. Louis encephalitis, of North America.

These viruses are known to infect a variety of domestic animals, often without producing symptoms. The distribution of antibody against the virus in the sera of animals has provided valuable information about the spread of viruses in America and Japan. Sera from a considerable number of animals in the Shepparton district, most of them from the immediate environment of patients suffering from encephalitis, have been tested by the complement-fixation test.

Sera of 53 sheep, 17 cows and heifers, five ducks and 16 pigs from north-eastern Victoria were found not to contain antiviral antibody, but 11 of 19 horses and two of four dogs exhibited complement-fixing antibody. At least in the case of horses this can be accepted as good presumptive evidence that the animal is commonly infected with the virus in the north-eastern part of Victoria. It may be that the virus only rarely causes clinical signs of illness in the horse. It is highly probable, but as yet unproven, that both man and the horse are infected by mosquitoes, but the full natural history of the disease has yet to be elucidated. It may be that horses and possibly dogs represent the important reservoir of virus from which human infection occasionally results. On the other hand, it is perhaps more likely that the primary reservoir of infection is to be found elsewhere (? in wild birds) and that horses play no significant part in the chain of events leading to human infection.

The identity of the Murray Valley encephalitis virus has been investigated serologically. Dr. J. L. O'Connor, of the Commonwealth Serum Laboratories, kindly made available a guinea-pig serum prepared by him against Japanese encephalitis B. This reacted strongly in a complement-fixation test with Murray Valley encephalitis virus. Although this result must be interpreted with caution, it is tentatively assumed that Murray Valley encephaliis virus is identical with or closely related to the virus of Japanese encephalitis B. The other properties of the Murray Valley encephalitis virus and the clinical and epidemiological characters of Murray Valley encephalitis are in complete agreement with this assumption.

The study of this virus and the clarification of its local epidemiology are far from complete, but the following conclusions may be stated provisionally.

1. A relatively small outbreak of encephalitis with a high mortality and due to the newly isolated virus has occurred in the Murray Valley area.

[1] The Director of the Walter and Eliza Hall Institute of Medical Research has asked for the publication of the accompanying account of investigations into an outbreak of encephalitis in the Murray Valley area. The investigations were initiated by Dr. S. G. Anderson and the virus was first isolated by Mr. E. L. French. As soon as the importance of the matter was recognized the cooperation of the Victorian State Health Department was sought and a larger team organized, including on the laboratory side Mrs. Margaret Donnelley and Miss Frances MacDonald, of the Hall Institute, and on the epidemiological and clinical side Dr. N. J. Caldwell, of the Victorian Department of Health, Mr. W. Gee, of the Victorian Department of Agriculture, Dr. H. McLorinan, Medical Superintendent, Fairfield Hospital, Dr. E. Graeme Robertson, Dr. W. Stevenson, of the Victorian Department of Health, and Mr. J. White, of Fairfield Hospital. This team has enjoyed the full cooperation of the staffs of the many hospitals involved.—Editor.

2. The virus is identical with or closely related to that of Japanese encephalitis B, and, like this, is probably carried by mosquitoes.

3. Horses and dogs show evidence of having been naturally infected.

4. The encephalitis virus has no relation whatever to myxomatosis in rabbits.

Reference.

Cleland, J. Burton, Campbell, Alfred W., and Bradley, Burton (1917), "The Australian Epidemics of Acute Polio Encephalomyelitis (X Disease)", Report of the Director-General of Public Health, New South Wales, for the year ended December 31, 1917.

Hospitals.

REPATRIATION GENERAL HOSPITAL, CONCORD.

STUDENTS commencing their study of clinical medicine at the Repatriation General Hospital, Concord, New South Wales, were addressed by Dr. George Bell, President of the Royal Australasian College of Surgeons.

In his address, Dr. Bell said that one of the earliest addresses to students entering hospital which he could remember had been delivered at Sydney Hospital by that distinguished surgeon and soldier, the late Dr. Thomas Fiaschi, some forty years previously. Dr. Fiaschi had exhorted the students to be diligent in their clinical studies, otherwise they would become filled with a "frothy vacuity". He told them that among their ranks there might perchance be a Laennec.

Dr. Bell went on to speak of René Théophile Hyacinthe Laennec, who had lived from 1781 to 1826 and was a Breton. It was interesting to note, he said, that Laennec's uncle, Guillaume François, who was the professor of medicine and *materia medica* at the University of Nantes, had been a pupil of John Hunter, the famous English anatomist. Laennec was a pupil of Jean Nicolas Corvisart, a great clinical teacher, who went round the wards with his students at the Charité Hospital in Paris examining each patient with the utmost care; "training of the senses" was his watchword. When he was only twenty-one years of age—about the age of the students listening—Laennec had written the first satisfactory description of peritonitis. He might be called the father of auscultation, and Sir William Osler had stated that with the stethoscope and an accurate study of disease at the bedside and in the post-mortem room he had almost created clinical medicine as it was known at the present day. There was an interesting story about his discovery of the stethoscope. Laennec had an exceedingly stout patient and could not hear the heart sounds satisfactorily by direct auscultation—in other words, by placing his ear against the patient's chest. On his way to see his patient he passed some children in the courtyard of the Louvre playing near a heap of timber. He noticed one child with his ear against the end of a small beam, listening to a signal transmitted to him by another child tapping the other end of the beam. Laennec immediately after that listened to the sounds in his patient's chest by using a quire of paper rolled into a cylinder, and he was able to hear the sounds more clearly.

Dr. Bell said that he hoped that it might be the privilege of his listeners to add, like Laennec, something to the art and science of medicine. He reminded them that they, as students, were pilgrims setting out on a voyage of discovery, which would last as long as they were doctors. *Ex Africam aliquid semper novum.* With the knowledge they had acquired in their pre-clinical studies they were entering now the most interesting period of their medical curriculum and beginning the study of disease at the bedside—and in the out-patient department—in living, sentient human beings.

Centuries before the Latin poet, Horace, had written:

> *Pallida mors aequo pulsat pede pauperum tabernas Regumque turres.* (Horace, Odes I, Carmen IV.)
>
> Pale death with impartial step knocks at poor men's cottages
> And palaces of kings.

Dr. Bell said that he liked to think of the students as the recruits of that army of medical men and women which was the implacable enemy of disease and death. As Fiaschi had remarked, "Disease knows no holiday". It would be their duty to prevent disease, or to seek it out and cure it.

They must never forget that the patient was a fellow human being, often in pain, mental and physical, and therefore they should be always gentle, courteous, considerate and truly charitable in their dealings with him. A patient should never be regarded simply as a case.

Dr. Bell exhorted them to be diligent and patiently observant, to study and examine each individual patient in detail. That was the only way in which they would acquire sound clinical knowledge and wisdom. They would notice that he emphasized the importance of self-help. The patient should be their book, just as the ground was the book of the Australian aboriginal or black-tracker. Printed books and journals had their uses, but they should not be placed first. Books alone were not enough. Sir William Osler had written: "To study the phenomenon of disease without books is to sail an uncharted sea—while to study books without patients is not to go to sea at all." The late Professor J. T. Wilson and the late Dr. Archie Aspinall had been fond of quoting: "If books ever become to the student a final and sufficient external authority for ascertainable fact, he will lose the impetus of the quest after, and his vision of, reality."

It was also important not to take diagnostic short cuts. Mechanical aids, X-ray examinations and pathological and biochemical investigations were often essential, but should not take first place. The findings must be correlated with the clinical history and clinical examination. They should be good listeners, particularly to the mothers of young children. The most able diagnosticians Dr. Bell had met had acquired their almost uncanny wisdom only by much patient and detailed examination at the bedside and in the post-mortem room and laboratory. By constant careful examination of patients, the students would acquire a sound knowledge of what was normal and would more readily recognize the abnormal.

Dr. Bell pointed out that in the present state of ignorance, the cure of all disease was not possible, but often both physical and mental suffering might be alleviated. He urged the students to respect the confidence of their patients and not to forget the high ideals of the Hippocratic oath: "Whatever in connection with my professional practice, or not in connection with my professional practice, I see and hear in the life of man which ought not to be spoken abroad—I will not divulge, as reckoning that all such shall be kept secret. . . ." They also had a duty to their teachers and to the nursing profession, to be courteous and helpful to them at all times. It was very important that they should be punctual in their attendance, take their clinical notes carefully and write them legibly.

In conclusion, Dr. Bell said that, having addressed them on their duty to the patient, the doctor and the nurse, he would remind them that they owed it to themselves not to neglect happy and healthful recreation, particularly in the fresh air. The motto *"Mens sana in corpore sano"* was an excellent one for medical folk, and particularly for medical students, to bear in mind. He wished them well with their studies and in the profession which they had chosen to follow.

Correspondence.

GENERAL PRACTICE TODAY IN URBAN AREAS.

SIR: As a rule, medical journals are mediums for the dissemination of highly technical information for the benefit of members of the medical profession; they contain nothing which could be of the least interest to the layman in his week-end reading. But the Jubilee Number of THE MEDICAL JOURNAL OF AUSTRALIA is in an entirely different category. The series of articles have a catholicity about them which provides absorbing reading for medical practitioner and layman alike, and I congratulate you, sir, on the production.

The Jubilee Number was brought to my notice because of a paragraph which was contained in one of the articles. The article, which was from the pen of Dr. Leigh Cook, of Claremont, Western Australia, was entitled "General Practice Today in Urban Areas". The particular paragraph to which my attention was invited amounted to a direct criticism of the Repatriation Department, and although I have no doubt that the criticism was made in good faith, I feel that the paragraph may create a wrong impression in the minds of your readers. For that reason it is incumbent on me to endeavour—through your columns, if I may—to clarify the position.

The paragraph in question was in the following terms:

> The work of the Repatriation Department has increased considerably in the post-war years. In spite of the form-filling required, doctors undertook this work cheerfully at concession rates after the first World War. The failure of the Repatriation Department to respond to repeated requests for any upward review of those rates and the fact that so many doubtful cases have been admitted to these benefits have resulted in nation-wide dissatisfaction among doctors.

I note the reference to "form-filling". This is a popular gibe which is levelled at the Public Service throughout the world and is generally based on an ill-founded prejudice. It will be found that "form-filling" takes place in all cases where government departments or companies or firms conducted by private enterprise have dealings with the public. Up-to-date business periodicals frequently mention the importance of "forms" in the conduct of business. They are just as essential in the business of a government department.

As regards the charge that the department had failed to respond to repeated requests for an increase in the rates paid to those practitioners who have been acting as repatriation local medical officers, I want to make it quite clear that the department was in no way responsible for any delay. On the contrary, the department has been endeavouring for some years to obtain increased rates for these medical officers, but because the launching of the National Health Scheme was believed to be imminent, the department was given to understand that the time was inopportune to consider any variation. You, sir, are well aware of the vicissitudes of the National Health Scheme, and there is no need for me to traverse the ground in this letter. It is sufficient for my purpose to say that the department never at any time allowed the question of increased rates for repatriation local medical officers to lapse. When the Government did eventually approve of a flat rate of 10s. a visit *plus* a mileage allowance in certain circumstances, the department took immediate steps to give effect to the decision as from the date thereof—December 12, 1950. Under date of December 19, 1950, I personally wrote to the General Secretary of the Federal Council of the British Medical Association informing him of the decision.[1]

I come now to the last charge made in the paragraph under review—the alleged dissatisfaction among doctors throughout the Commonwealth that so many doubtful cases have been admitted to medical benefits under the *Repatriation Act*. I fully realize that a busy medical practitioner has not the time, even if he had the inclination, to make himself familiar with the various provisions of the repatriation legislation; but it is reasonable, I think, to expect that one who writes for publication should satisfy himself as to the correctness of comments which he may contemplate about a public department. The words in the paragraph imply that the various statutory authorities—the Repatriation Commission, the Repatriation Boards, and the Appeal Tribunals—in admitting doubtful cases to benefits are a law unto themselves.

This implication is untrue, to say the least, and although I have already taken up much of your space, sir, I crave your indulgence still further by permitting me to quote Section 47 of the *Repatriation Act*, 1920-1950. The section reads:

> 47.—(1.) The Commission, a Board, an Appeal Tribunal and an Assessment Appeal Tribunal, in hearing, determining or deciding a claim, application or appeal, shall act according to substantial justice and the merits of the case, shall not be bound by technicalities or legal forms or rules of evidence and shall give to the claimant, applicant or appellant the benefit of any doubt—
>
> (*a*) as to the existence of any fact, matter, cause or circumstance which would be favourable to the claimant, applicant or appellant; or
>
> (*b*) as to any question whatsoever (including the question whether the incapacity from which the member of the Forces is suffering or from which he has died was contributed to in any material degree, or was aggravated, by the conditions of his war service) which arises for decision under his claim, application or appeal.
>
> (2.) It shall not be necessary for the claimant, applicant or appellant to furnish proof to support his claim, application or appeal, but the Commission, Board, Appeal Tribunal or Assessment Appeal Tribunal determining or deciding the claim, application or appeal shall be entitled to draw, and shall draw, from all the circumstances of the case, from the evidence furnished and from medical opinions, all reasonable inferences in favour of the claimant, applicant or appellant, and in all cases whatsoever the onus of proof shall lie on the person or authority who contends that the claim, application or appeal should not be granted or allowed to the full extent claimed.

It will thus be seen that before a claim, application or appeal can be refused, the Repatriation Commission or a Board must discharge the onus of proof. This can, I feel, be best explained by quoting the following extract from the judgement of Mr. Justice Starke in the High Court of Australia in what is known as "Bott's Case" (50 C.L.R., page 255), which is still applicable to the question of discharging the onus of proof:

> Suppose, however, after considering the nature and strength of the proofs offered in support or denial of the main fact to be established the Appeal Tribunal is left in doubt as to which way it should decide that fact, then the Section directs that the appellant shall be given the benefit of the doubt, or in effect, enacts that in such circumstances the Commission has failed to satisfy the burden of proof cast upon it.

That is the law on the subject, and the various authorities are carrying out that law, and thus giving effect to the will and intention of the Parliament.

Yours, etc.,

Walter J. Cooper,
Minister for Repatriation.

Brisbane,
March 20, 1951.

[1] By the beginning of December the whole of the Commonwealth Jubilee Number had been printed.—Editor.

THE PROBLEM OF THE THORACIC STOMACH.

Sir: In your issue of March 17, 1951, an article by Dr. A. W. Morrow entitled "The Problem of the Thoracic Stomach" draws attention to various lesions which occur in the region of the œsophageal hiatus of the diaphragm. Some of the points made by Dr. Morrow are worthy of emphasis if ill-advised and misdirected operations are to be avoided. In particular I would like to draw attention to the important contributions to this subject made in England, especially by Allison and Johnstone, of Leeds. The following views are representative of thought in England on this problem:

1. Professor Johnstone, whilst examining many hundreds of patients undergoing barium meals, has been able to demonstrate frequently that considerable laxity of the hiatus can exist, and that portion of the stomach can be made to herniate into the chest, especially in patients of the age and build that Dr. Morrow has stressed. These lesions are asymptomatic in themselves, and patients are not informed of their presence. This condition, in its gross forms, is called an œsophago-gastric hernia by Akerlund, and is virtually a "sliding" type of hernia. Reflux of gastric juice into the lower end of the œsophagus is liable to occur.

2. The truly short œsophagus associated with a "thoracic stomach" is nearly always an acquired lesion which follows scarring of the lower end of the œsophagus. This scarring may be due to reflux of gastric juice with resultant œsophagitis and ulceration, or it may follow peptic ulceration of the œsophagus occurring in areas of ectopic gastric mucosa. The accompanying hernia is secondary to these changes.

The evidence presented by Allison and Johnstone for the above changes is based on long and careful work and is most convincing. If one accepts their views it is apparent that many operations aimed at the correction of the hernia have been misconceived in that the patient was suffering from œsophagitis, peptic ulceration or a stricture of the œsophagus. The repair of such a hernia can be difficult, or impossible, and the patient is either unrelieved or made worse.

The paraœsophageal hernia, on the other hand, is a different condition. Laxity of the hiatus permits herniation of the stomach (and other viscera occasionally), the œsophagus being of normal length. Such a hernia may be complicated by inflammation or strangulation. It is essential that this condition should be distinguished from the case with a short œsophagus. At times this may be difficult on clinical and radiological grounds alone, and in such cases œsophagoscopy may be necessary to identify the œsophago-gastric junction, with biopsy of the mucous membrane if in doubt.

Although the distinction between these two types of hiatus hernia is not quite as clear cut as I have indicated, this concept renders diagnosis and management more rational. Treatment, in England, is usually along the lines indicated by Dr. Morrow. However, I would question the statement that in the younger group of patients "resection of the stricture is probably preferable". Advocacy of this means bringing the stomach up into the chest, or using one of the methods of œsophago-jejunostomy. The procedure of œsophago-gastrostomy at best is liable to be complicated by minor degrees of ill-health, and at worst by a recurrence of the condition for which the operation has been performed. In the present state of our knowledge, dilatation, when possible, still remains the best form of treatment for a non-malignant stricture of the œsophagus.

Dr. Morrow's advocacy of conservatism is timely, but I feel is overstressed. He has given a list of serious complications which can, and do, occur. I submit that a hiatus hernia may present the same fundamental problems as an inguinal hernia. The repair of such a hernia is technically easy and attended by a minimal morbidity and mortality rate. The decision to operate on this condition should be guided by the same criteria as applied to any hernia.

Yours, etc.,

IAN MONK.
Thoracic Unit,
The Royal North Shore Hospital of Sydney,
Crows Nest,
New South Wales.
March 20, 1951.

A MORE REALISTIC VIEW OF TUBERCULOSIS.

SIR: It is now some months since my letters on this subject were published in the journal (February 18 and May 27, 1950). The replies to these letters showed that I had failed to make my case and attitude sufficiently clear to some correspondents. In order to prevent any future misunderstanding, I will explain that my acquaintance with the administration and institutional side of tuberculosis had been mainly with the Repatriation Department and that this association had first aroused my interest and criticism on this subject.

In the first place, I will again state my profound scepticism as to the necessity of "institutionalizing" for adequate investigation these symptomless and apparently healthy adults who are found to have some evidence suggestive of a minimal pulmonary tuberculous lesion on routine radiological examination. It seems a strange paradox that we should put to bed (or at least drastically restrict their activities) apparently healthy people to make them "better". It is difficult to appreciate the reason of such treatment. I can think of no other infectious disease in which it is considered necessary to prescribe rest presumably in order to restore them to the health they are already enjoying. It is hard to imagine how their resistance is to be increased by an enervating regime which will subsequently require a hardening-up process by graduated stages to restore them to the same vitality from which they have been reduced by this meddlesome interference.

No one will deny that those symptom-free people who show X-ray evidence of a minimal tuberculous lesion are more likely to have tuberculosis and become ill with it than those who have no such evidence. Such people require some investigation and follow-up, but surely this investigation should be as out-patients and be limited and not too frequent. It should not interfere with their earning a living (except in a few proscribed industries) and should not under any circumstances turn them into "privileged pensioners" for life on the strength of a shadow that could be sometimes covered by a shilling piece. There would not be such a real shortage of beds for the real sufferers of tuberculosis, who often deteriorate hopelessly in unsuitable accommodation at home, or in lodgings, while beds are being occupied by the privileged ex-service personnel who have been admitted for reasons that savour more of politics than medicine.

I am indebted to one of my critics, Dr. Eric Clarke (THE MEDICAL JOURNAL OF AUSTRALIA, June 24, 1949), for introducing me to the Propit Survey of Tuberculosis in Young Adults. I have not yet completed a thorough examination of this survey, but on page 163, paragraph 2, it states: "The subject of the small Tuberculosis lesion is one of growing importance. It is generally agreed that treatment of Tuberculosis at an early stage offers greater prospects of final cure than treatment at more advanced stages. ***It is still not known what proportion of small lesions heal spontaneously*** (the italics are my own).

At the Australasian Medical Congress held in Brisbane during last year, and reported in THE MEDICAL JOURNAL OF AUSTRALIA of July 15, 1950, speakers discussed the minimal tuberculous lesion. Dr. Cotter Harvey quoted the Propit Survey and then continued by saying that in mass surveys, three-quarters of those found to have pulmonary tuberculosis had minimal lesions; untreated, 25% of these lesions would progress within six to twelve months, but with prompt treatment 90% would be permanently arrested. Authority for Dr. Harvey's statement as regards the permanent arrest in 90% of cases treated early is not obvious. It would appear that he was combining the results of the Propit Survey with the work of Amberson (1937) also mentioned in the Propit Survey. If this is so, Dr. Harvey is indulging in a very doubtful basis of comparison. He is not only multiplying the margin of error by comparing two sets of figures from different sources, but he is combining two surveys which are not comparable.

Dr. A. H. Penington gave his opinion that for patients with minimal lesions found during routine X-ray examination, when the time of the onset of the lesion was not known, it was wise to keep them under close observation and if possible *to allow them to lead a normal life* (the italics are my own).

If we are to accept Dr. Harvey's figures, the minimal lesions should be treated in an institution. On the other hand, if we agree with Dr. Penington, close observation only is necessary. Neither of these authors implied any discrimination concerning the minimal lesions as to whether they were accompanied by symptoms or not.

Yours, etc.,

B. SHORT.
Cessnock,
New South Wales,
Undated.

BLOOD ADRENALINE CONTENT IN PINK DISEASE.

SIR: During Professor Sir Stanton Hicks's eight months' absence from the Department of Physiology and Pharmacology, Dr. D. B. Cheek was given facilities for estimation of blood adrenaline of children, both normal and with pink disease. It has to be stated without commentary that the undersigned, who was in charge of the department during this time, has not authorized Dr. Cheek to publish from this department any of these findings, which, at this stage, were considered as only preliminary. Consequently, the above department does not take any responsibility as to critical evaluation and presentation of the figures obtained and as to conclusions drawn from them.

Yours, etc.,

F. LIPPAY, M.D., D.Sc.
Department of Physiology and Pharmacology,
University of Adelaide,
Adelaide.
March 16, 1951.

THE HOSPITAL ALMONER PROBLEM.

SIR: May I express my appreciation of your editorial of January 20, stressing the need for more almoners in Australia.

Although a very small hospital, Rachel Forster Hospital was one of the first hospitals in Australia to have an almoner department, and over the years has been extremely fortunate in its almoner staff, which has helped in no small way to build up the reputation of the hospital for service to the community.

Whilst the need for trained almoners has grown, the number of almoners available has dropped, creating a serious gap in the service rendered by hospitals to the public.

The work is there, but not the trained personnel to carry it out.

Yours, etc.,

MARY C. PUCKEY,
Chief Executive Officer and
Medical Superintendent.
The Rachel Forster Hospital for Women and Children,
Pitt Street,
Redfern,
New South Wales.
March 19, 1951.

REPORT OF TWO CASES OF HERPES ZOSTER TREATED WITH "CHLOROMYCETIN".

SIR: THE MEDICAL JOURNAL OF AUSTRALIA, March 3, 1951, contains a report of two cases of *herpes zoster* treated with "Chloromycetin", with apparent good result in one and an amazing improvement in the other. For those unacquainted with the natural history of the disease, it is apposite to point out that the eruption of *herpes zoster* not uncommonly settles spontaneously within a week to ten days. It is impossible to predict the course of the disorder. There is little relationship between pain, irritation, duration and extent of the eruption. If enthusiasm could be maintained after treatment of many cases with an adequate control series, it would mean much to dermatologists, who are continuously facing the problem of this capricious disorder. It is felt that the cases reported may not have been affected in any way by the use of this drug.

Yours, etc.,

IAN O. STAHLE,
Dermatologist, Royal Melbourne Hospital.

110 Collins Street,
Melbourne,
March 21, 1951.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Week-End Course at Wagga Wagga.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that a week-end course will be held at the Wagga Wagga Base Hospital, in conjunction with the Southern Districts Medical Association, on Saturday and Sunday, April 14 and 15, 1951. The outstanding feature of the course this year is the inclusion of Dr. William Pickles, M.D., M.R.C.P., the well-known Yorkshire general practitioner, who is at present on a lecture tour of Australia.

The programme will be as follows:

Saturday, April 14: 2 p.m., registration; 2.30 p.m., "Research in General Practice", Dr. William Pickles; 4 p.m., "The Painful Shoulder", Dr. Carlyle Hudson.

Sunday, April 15: 10 a.m., "Gynæcological Endocrinology", Dr. Bruce Williams; 11.30 a.m., "Epidemiology in Country Practice", Dr. William Pickles; 2 p.m., "Obstetrical Difficulties", Dr. Bruce Williams; 3.30 p.m., "Backache", Dr. Carlyle Hudson.

The fee for attendance is £2 2s. Those wishing to attend are requested to notify Dr. J. S. Storey, Honorary Secretary, Southern Districts Medical Association, 69 Baylis Street, Wagga Wagga (telephone 2568), as soon as possible.

Clinical Meeting at Balmoral Naval Hospital.

The date of the clinical meeting at the Balmoral Naval Hospital, Balmoral, in April has been changed from April 17 to Tuesday, April 24, 1951. At this meeting Dr. T. M. Greenaway will speak on "Some Aspects of 'Functional' or Psychosomatic Diseases" at 2 p.m. Clinical cases will be shown at 4 p.m.

The meeting is open to all medical practitioners.

Week-End Course at Katoomba.

The Post-Graduate Committee in Medicine in the University of Sydney announces that, in conjunction with the Blue Mountains Medical Association, a week-end course will be held at Katoomba, in the ballroom of the Carrington Hotel, on Saturday and Sunday, April 21 and 22, 1951. The programme is as follows:

Saturday, April 21, 1951: 2 p.m., registration; 2.30 p.m., "Diabetes", Dr. Kenneth T. Hughes; 4 p.m., "Causes of Stillbirths and Neo-Natal Deaths", Dr. Mary Heseltine.

Sunday, April 22, 1951: 10 a.m., "Treatment of Chronic Ulceration of the Legs", Dr. John Loewenthal; 11.30 a.m., "Recent Advances in Clinical Medicine", Dr. Kenneth T. Hughes; 2.30 p.m., "Useful Laboratory Investigations Associated with Obstetrics and Gynæcology", Dr. Mary Heseltine; 4 p.m., "Anticoagulants and Antibiotics", Dr. John Loewenthal.

The fee for attendance is £2 2s. Those wishing to attend are requested to communicate as soon as possible with Dr. Nicholas Larkins, Honorary Secretary, Blue Mountains Medical Association, Katoomba Street, Katoomba.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Numbers 19 and 20, of March 15 and 21, 1951.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Promotion.—Surgeon Lieutenant-Commander Kenneth Charles Armstrong is promoted to the rank of Surgeon Commander, dated 2nd December, 1950.

Royal Australian Air Force.

Active Citizen Force: Medical Branch.

Flight Lieutenant A. T. Pearson (022807) is transferred to the Reserve, 30th January, 1951.

Air Force Reserve: Medical Branch.

The following former officers are appointed to commissions with the ranks indicated: (Temporary Wing Commander) W. A. Seldon (261220), 6th December, 1950. (Flight Lieutenant) J. B. Felstead (257109), 1st November, 1950.

The following are appointed to commissions with the rank of Flight Lieutenant: George Montario Bedbrook (257798), 29th November, 1950; Jacob Zimmet (287474), 6th December, 1950.

The appointment of Flight Lieutenant J. H. Wall (266333) is terminated, 20th October, 1950.

Obituary.

DOUGLAS LEWIS BARLOW.

THE sudden death of Dr. Douglas Lewis Barlow on December 4, 1950, has already been recorded in these pages. To his friends it had been apparent for some time that he was not in normal health. It was hoped that a business and holiday trip to England would help, but on his return to Australia last September grim portents were evident.

Douglas Barlow began his education at Prince Alfred College, Adelaide, where he was *dux* and Elder Scholar in 1910. In 1915 he graduated as Bachelor of Medicine and Bachelor of Surgery at the University of Adelaide and enlisted on graduation in the Australian Imperial Force. He became regimental medical officer of the Seventh Battalion and was awarded the Military Cross at Ypres in 1917. He continued his close association with military work and was for some years Lieutenant-Colonel in command of the Sixth Cavalry Field Ambulance. Eventually he was transferred to the Retired List with the rank of colonel.

After the 1914-1918 war Barlow did post-graduate work in England and on his return to Australia took, in 1922, the degree of Doctor of Medicine of the University of Adelaide. He held several appointments during the 1920's. He was honorary pathologist and bacteriologist at the Adelaide Children's Hospital in 1922 and 1923; he was honorary clinical pathologist at the Adelaide Hospital from 1921 to 1923, and demonstrator in pathology and bacteriology at the University of Adelaide during the same period. He visited Great Britain a second time in 1929 for post-graduate study, and it was from this time that he turned his attention more particularly towards the study of allergy. From 1930 onwards he was honorary medical officer at the asthma clinics of the Adelaide Children's Hospital and the Royal Adelaide Hospital. He was a foundation Fellow of The Royal Australasian College of Physicians and in 1947 was elected a Fellow of the International Association of Allergists.

In a personal appreciation of Douglas Barlow, Dr. R. A. Haste has written that golf and tennis were his major loves. Over many years he and his wife generously entertained colleagues and their friends on his beautiful tennis court. In latter years he became a keen fisherman. He owned a fishing shack on Yorke Peninsula and with his family and his friends spent many happy days there. His colleagues, who will miss his genial personality and his special knowledge of allergic conditions, extend their sympathy to his wife, his daughter (a medical practitioner) and two sons.

CHARLES ERNEST SANDFORD JACKSON.

WE regret to announce the death of Dr. Charles Ernest Sandford Jackson, which occurred on March 25, 1951, at Cloncurry, Queensland.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1950, of New South Wales, as duly qualified medical practitioners:

Cooper, John Giffard Brassey, M.B., B.S., 1947 (Univ. Melbourne), 27 Heygarth Street, Echuca, Victoria.

Glick, Myer, M.B., Ch.B., 1936 (Univ. Leeds), M.R.C.S. (England), L.R.C.P. (London), 1937, F.R.C.S. (England), 1948, 155 Bellevue Road, Bellevue Hill.

Nally, John Brendan Aloysius, L.R.C.P., L.R.C.S. (Ireland), 1948, Waterfall Sanatorium, Waterfall.

Navratil, Deszo, M.D., 1900 (Univ. Budapest); also recommended and approved for registration in terms of Section 17 (2) of the *Medical Practitioners Act*, 1938-1950, 209 Victoria Street, King's Cross.

O'Brien, Richard Alfred, M.B., B.S., 1902, M.D., 1909 (Univ. Melbourne), 76 Banksia Street, Bowral.

Webster, Jessie Charlton, M.B., B.S., 1949 (Univ. Melbourne), c.o. Bernly Private Hotel, Potts Point.

The following additional qualification has been registered:

Simpson, Ian Gordon, 73 Alt Street, Ashfield (M.B., B.S., 1942, Univ. Sydney), D.P.M., 1949 (Univ. Sydney).

TASMANIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Gregory, Lee Westbrook, M.B., B.S., 1949 (Univ. Queensland), Launceston General Hospital, Launceston.

Reid, Colin Stuart, M.B., B.S., 1949 (Univ. Melbourne), Royal Hobart Hospital, Hobart.

Ciezar, Mieczyslaw George, M.D. (Univ. Warsaw), Department of Health, Hobart.

Bennett, Winfield Robert Curtis, M.B., B.S., 1950 (Univ. Melbourne), Derby.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts*, 1939 *to* 1948, of Queensland, as duly qualified medical practitioners:

Pryor, Gordon Alfred William, M.B., B.S., 1944 (Univ. Melbourne), Mount Isa Hospital, Mount Isa.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED MARCH 10, 1951.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Ankylostomiasis	•	..	1	..	..	..	..	..	1
Anthrax	•	..	..	..	..	..	..	..	..
Beriberi	•	•	..	•	•	•	..	•	..
Bilharziasis	•	..	..	..	..	1(1)	..	..	1
Cerebro-spinal Meningitis	..	..	1	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(*a*)	•	•	..	•	•	•	•	•	..
Dengue	•	•	•	•	•	•	..	•	..
Diarrhœa (Infantile)	..	..	20(18)	..	3(2)	..	..	..	23
Diphtheria	3(2)	1(1)	2(1)	..	8(6)	..	..	..	19
Dysentery (Amœbic)	•	..	..	..	..	..	..	..	..
Dysentery (Bacillary)	•	3(3)	..	..	6	..	..	..	9
Encephalitis Lethargica	..	..	1	..	..	..	..	..	1
Erysipelas	•	•	•	2(1)	•	•	..	..	2
Filariasis	•	..	..	..	..	..	..	..	..
Helminthiasis	•	..	•	•	•	•	•	•	..
Hydatid	•	..	•	•	..	•	•	..	..
Influenza	•	•	•	..	•	•	..	•	..
Lead Poisoning	•	•	..	..	•	•	•	•	..
Leprosy	..	..	..	..	1	..	..	..	1
Malaria(*b*)	•	..	1	..	..	..	..	..	1
Measles	•	•	•	23(4)	•	•	..	..	23
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	52(17)	4(2)	28(5)	26(11)	..	10(2)	..	..	120
Psittacosis	•	..	•	..	•	•	•	•	..
Puerperal Fever	..	..	1	..	2	1	..	..	4
Rubella(*c*)	•	•	..	•	..	•	..	..	..
Scarlet Fever	9(5)	11(6)	4(1)	2(1)	6(3)	1	..	..	33
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	•	..	..	•	•	•	•	•	..
Trachoma	•	..	..	•	•	•	•	•	..
Tuberculosis(*d*)	47(35)	15(9)	32(8)	3(3)	12(5)	2(2)	..	..	111
Typhoid Fever(*e*)	1	..	1	..	1(1)	..	..	..	3
Typhus (Endemic)(*f*)	..	..	..	..	..	..	..	..	..
Undulant Fever	..	..	..	..	..	..	..	..	..
Weil's Disease(*g*)	•	•	3	•	•	•	•	•	3
Whooping Cough	•	•	•	2(2)	•	•	..	..	2
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 37, 1946–1947. Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from the Northern Territory.
• Not notifiable.
(*a*) Includes Mossman and Sarina fevers. (*b*) Mainly relapses among servicemen infected overseas. (*c*) Notifiable disease in Queensland in females aged over fourteen years. (*d*) Includes all forms. (*e*) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (*f*) Includes scrub, murine and tick typhus. (*g*) Includes leptospiroses, Weil's and para-Weil's disease.

Brown, Kenneth James, M.B., B.S., 1950 (Univ. Sydney), Longreach, Queensland.

Lekias, John Simon, M.B., B.S., 1946 (Univ. Queensland), 203 Lake Street, Perth, Western Australia.

The following additional qualification has been registered:

Watson, Donald, 14 Circe Street, Hamilton, Brisbane (M.S., 1950, Univ. Queensland).

Notice.

PSYCHIATRY AND RELIGION.

THE Williams Foundation is again sponsoring a programme of lectures this year. The opening lecture will be delivered by the Reverend Dr. Leslie D. Weatherhead, M.A., D.D., Ph.D., on the subject of "Psychology, Religion and Healing" in the Assembly Hall, Collins Street, Melbourne, on Friday, March 30, at 8 p.m.

Reserved seats at Glen's six days in advance, 4s.; unreserved, at the door, 2s.

Subsequent to Dr. Weatherhead's lecture, Dr. W. L. Carrington and the Reverend Frank Borland, M.A., will give six lectures on "The Science and Art of Human Relationships", taking alternate lectures. Dr. Carrington starts with "The Modern Approach to Human Relationships", after which the Reverend Frank Borland deals with "The Value, Limitations and Future of Social Science". Then follow "The Social Biology of the Family", "Clinical Approaches to Social Pathology", "Education for Marriage and Parenthood", and "The Way to Social Health and World Peace". The lectures will be given in the Anatomy Theatre, University of Melbourne (enter from Swanston Street), at 8 p.m. on Tuesday nights, April 10, 17 and 24 and May 1, 8 and 15. Admission is free and without ticket.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Perry, Grace Amelia, M.B., B.S., 1951 (Univ. Sydney), St. George Hospital, Kogarah.

Webster, Jessie Charlton, M.B., B.S., 1949 (Univ. Melbourne), 51 Milson Road, Cremorne.

Duncan, Glen Malcolm, M.B., B.S., 1951 (Univ. Sydney), 40 Princes Street, Mortdale.

The undermentioned has been elected a member of the New South Wales Branch of the British Medical Association:

Phillips, John Bertram, M.B., B.S., 1946 (Univ. Sydney), 20 Thomas Avenue, Roseville.

Congresses.

INTERNATIONAL CONGRESS OF PURE AND APPLIED CHEMISTRY.

THE Twelfth International Congress of Pure and Applied Chemistry will be held in New York City, United States of America, in September, 1951, in connexion with the seventy-fifth anniversary meeting of the American Chemical Society. The section on food, nutrition and agricultural chemistry will welcome research papers on food, nutrition and agricultural chemistry from all foreign research workers who plan to attend the New York meetings. Application forms and detailed information can be obtained by writing to Dr. Harry L. Fisher, Administrative Assistant, 2101 Constitution Avenue, Washington 25, D.C., United States of America, or to Dr. R. Adams Dutcher, Secretary, Section 6, Food and Nutrition, State College, Pennsylvania, United States of America.

Medical Appointments.

Dr. P. O. Flecker has been appointed government medical officer at Mareeba, Queensland.

Dr. J. L. D. Scott has been appointed a medical referee for the purposes of *The Workers' Compensation Acts*, 1916 *to* 1949, of Queensland.

Diary for the Month.

APRIL 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee. Organization and Science Committee.
APRIL 13.—Queensland Branch, B.M.A.: Council Meeting.
APRIL 17.—New South Wales Branch, B.M.A.: Medical Politics Committee.
APRIL 18.—Western Australian Branch, B.M.A.: General Meeting.
APRIL 19.—New South Wales Branch, B.M.A.: Clinical Meeting.
APRIL 19.—Victorian Branch, B.M.A.: Executive Meeting.
APRIL 24.—New South Wales Branch, B.M.A.: Ethics Committee.
APRIL 25.—Victorian Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney)—All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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